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Archives of Neurology and Psychiatry

VOLUME 16 DECEMBER, 1926 NUMBER 6

STUDIES ON THE CENTRAL VISUAL SYSTEM

IV. THE DETAILS OF THE ORGANIZATION OF THE GENICULOSTRIATE SYSTEM IN MAN *

TRACY JACKSON PUTNAM, M.D.

BOSTON

In the preceding papers of this series,[1] evidence for the following propositions was adduced: The projection of the retinal quadrants and the macula on the human striate cortex can be determined from purely anatomic considerations (normal, pathologic and experimental); the geniculostriate radiation occupies the whole of the fasciculus longitudinalis inferior (external sagittal stratum) and this alone; the upper retinal quadrant is represented superiorly in the cortex and radiation, mesially in the corpus geniculatum and, in the lower quadrant, inferiorly in the cortex and laterally in the geniculate body; the representation of the macula is extensive and lies in the posterior portion of the visual area; and the existence of callosal visual fibers is improbable.

Having made so much progress, we may be encouraged to attempt an answer to the following additional questions:

1. What is the extent of the representation of the macula in the striate cortex?

2. Where and how extensively is the macula represented in the fasciculus longitudinalis? Why is the quadrantic form so common in partial hemianopia?

3. To what degree do various groups of fibers (e. g., fibers from homologous points in the two retinas) change their relative positions in the fasciculus longitudinalis, in running from the geniculate body to the cortex?

4. What is the course of fibers between the compact fasciculus and the cortex? Why is there so often an interruption in tracing degenerations from radiation to cortex, and vice versa? Why are the fibers

* From the Central Institute for Brain Research, and the Neurological Laboratory of the Binnengasthuis, Amsterdam.

1. Putnam, T. J.; and Putnam, Irmarita: Studies on the Central Visual System: I. The Projection of the Retina on the Striate Cortex of the Rabbit, Arch. Neurol. & Psychiat. **16**:1 (July) 1926. Putnam, T. J.: II. A Comparative Study of the Form of the Geniculostriate Visual System of Mammals, ibid. **16**:263 (Sept.) 1926. III. The General Relationships in the External Geniculate Body, Optic Radiation and Visual Cortex, ibid. **16**:566 (Nov.) 1926.

of the fasciculus longitudinalis inferior sometimes reported to end in the striate area alone, sometimes also in the lateral surface of the occipital lobe?

Most authors who have concerned themselves with the organization of the visual system have relied for information on the study of sections alone. To find a more complete answer, it seemed desirable to utilize quantitative and also three-dimensional methods in their investigation, as far as practicable. The studies on which the present paper is based include (*a*) an estimation of the area and plane shape of the area striata; (*b*) an estimation of the area of cross-section of the fasciculus longitudinalis inferior; (*c*) an estimation of the volume of the corpus geniculatum externum; (*d*) a reconstruction of the normal central visual system from myelogenetic preparations and a pathologic case in which the radiation was isolated; (*e*) a reconstruction of the central visual system, in a case in which there were small degenerations in the cortex, fasciculus longitudinalis and corpus geniculatum, to show the course of the fibers in the radiation; (*f*) quantitative estimations of atrophic areas in the cortex, radiation, and corpus geniculatum in pathologic cases. By combining the results of these investigations with certain facts already accepted, some light is thrown on the whole problem.

SIZE AND PLANE SHAPE OF THE HUMAN AREA STRIATA

The size of the area striata was estimated in five apparently normal occipital lobes—four from adults and one from a new-born baby. Two of these—a frontal and sagittal series—were the "control" series of the Central Institute for Brain Research. Two were from a case of Wilson's disease, in which the visual system appeared unaffected. The fifth was from the same new-born child from which the reconstruction to be described was modeled.

The method was as follows:

Sections were selected throughout the series so as to be as nearly as possible a uniform distance—2, 3 or 4 mm.—apart. Thus, if the sections were 30 microns thick, every tenth one was taken. The sections were projected on a sheet of paper by means of a projection lantern with a magnification of 10. The ends and the course of the stripe of Gennari could be plainly seen. Sometimes it appeared in two or more isolated parts. The total length was measured by means of a so-called curvimeter or cartometer (used for measuring maps) divided by 10, and plotted on coordinate paper, at intervals corresponding to the intervals between the sections taken. The approximate depth of the calcarine fissure was similarly measured from one end or the other, and its course is marked by a series of crosses in the accompanying charts. Similarly, the size and position of degenerated areas could be determined and plotted.

Remeasurements of the same cortex on a different series of sample slides showed the mean error to be about 5 per cent. The area was estimated by counting the contained square millimeters.

The results of the estimation are seen in figures 1, 2, 3, 4 and 5. With them should be considered figure 19, which is the chart of the left

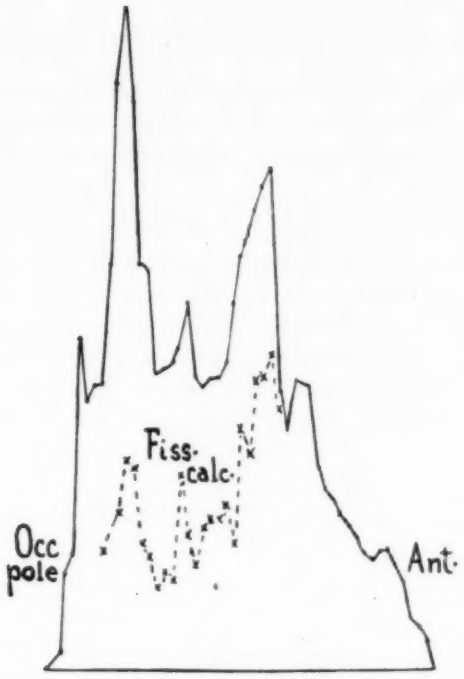

Fig. 1.—Plane map of the area striata (enclosed by solid line), from the left hemisphere of a case of Wilson's disease, in which the visual system appeared intact, cut in frontal series. The position and course of the calcarine fissure (*Fiss. calc.*) is indicated by a broken line and crosses. The total area of the visual cortex measures about 18.8 sq. cm. Natural size. (Method of construction is given in the text.)

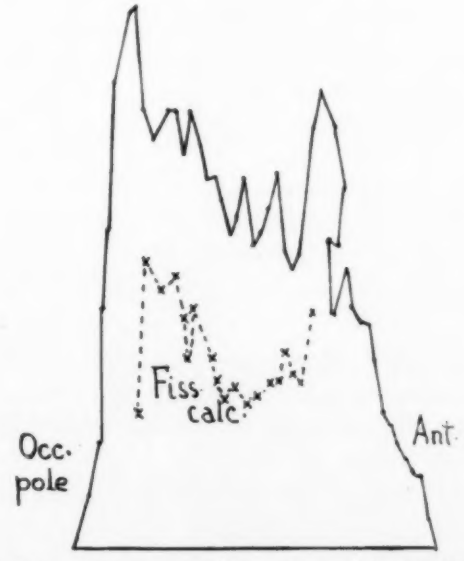

Fig. 2.—Plane map of the area striata of the right hemisphere of the same case as figure 1. Total area of the visual cortex, about 18.38 sq. cm. Natural size.

hemisphere of Brouwer's [2] case 2, used also for a reconstruction to be described. Here there was a lesion, but its size could readily be measured, as the termination of the stripe of Gennari could be made out in all but two sections.

Size of Striate Area.—The size and the limits within which it varies prove to be the most important points elicited. The smallest area was 18.3 sq. cm., the largest 24.57 sq. cm., the average 20.7 sq. cm. The size in each case appeared to maintain a certain proportion to the size of the brain as a whole; but this was unfortunately unknown in any of the series. The difference between the two "normal control" series (figs. 2 and 3), derived from hemispheres of the same brain, is perhaps

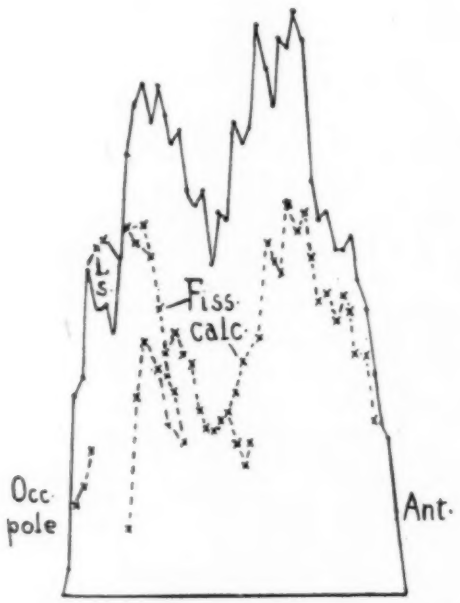

Fig. 3.—Plane map of the area striata in a normal frontal series. A small patch of visual cortex lies on the lateral surface (*L. s.*). The complicated course of the calcarine fissure (*Fiss. calc.*) should be noted. Total area, about 23.38 sq. cm. Natural size.

explained by the fact that there were so few sagittal sections that the percentage of error was greatly increased. The loss of only three slides would account for the discrepancy. The size of the baby's striate cortex does not seem surprising.

Shape of Striate Area.—The shape of this area is not very enlightening. In the rabbit's cortex, the location of the binocular field is marked

2. Brouwer, B.: Ueber die Sehstrahlung des Menschen, Monatschr. f. Psychiat. u. Neurol. **41**:129-158 and 203-234, 1917.

by an irregularity in the otherwise symmetrically oval striate area. Nothing so definite is to be made out in the human brain. Perhaps the most we can say is that the length of the stripe of Gennari usually decreases rapidly as the anterior end of the calcarine fissure is approached.

The vagaries of the calcarine fissure are of interest. It certainly does not divide the area accurately in two, and is an unreliable landmark for orientation. Usually more of the visual cortex lies below it than above. The striate cortex extends further anteriorly along its lower lip than along its upper lip—a fact that has long been recognized by neuro-anatomists, but which has led to great confusion, for example in regard to the articulation of the ventral edge of the fasciculus longitudinalis with the cortex. The horizontal (lower) limb of the fasciculus longitudinalis is sometimes said to be in connection with the anterior part of the cortex, sometimes with the inferior part. To a certain extent, both are true.

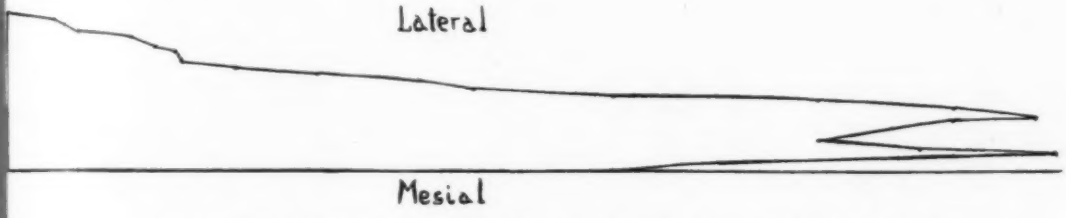

Fig. 4.—Plane map of the area striata in a sagittal series, from the other hemisphere of the same brain as figure 3. The construction is different from that of the other charts in the series. Instead of being laid out vertically beginning with the occipital pole, measurements have been laid out horizontally beginning with the mesial surface. This perhaps explains the discrepancy between the area of visual cortex in this hemisphere (18.5 sq. cm.) and that of the other, shown in figure 3. Natural size.

AREA OF CROSS-SECTION OF THE NORMAL FASCICULUS LONGITUDINALIS

The determination of the area of cross-section is attended by a number of difficulties. In the first place, the fasciculus is clearly outlined only (*a*) in myelogenetic preparations, (*b*) in the brains of some lower animals, (*c*) in pathologic cases in which there is a diffuse degeneration of the surrounding white matter and (*d*) in certain normal brains, under conditions of staining which are not well defined. The distinction between the coarse, dark fibers of the great projection systems and that of the remaining white matter is made clearer by prolonged bleaching with permanganate, and less clear by too long treatment with lithium carbonate. Other factors are involved, however, which would repay further study; a method of sharper differentiation between different

fiber systems in the normal adult brain might supplant the use of infants' brains to some extent.

In the second place, the plane of the section is of importance, as an oblique section would show a larger area than a correct transverse one. From the shape of the reconstructions to be described, however, it seems justifiable to conclude that the frontal section as usually cut, perpendicular to the flat surface on which the brain lies, will yield an approximate cross-section.

The measurement of the fasciculus in an appropriate section is not difficult. With a projection camera, it is drawn, enlarged ten times, on cross-section paper; either the figure is cut out and weighed, or the contained squares are counted. The average error appears to be about

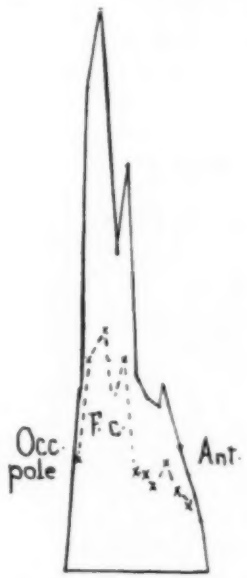

Fig. 5.—Plane map of the area striata of a new-born child. The proportion of it lying on the lateral surface is not shown. Area, 5.54 sq. cm. Natural size.

10 per cent. Usually four sections from each series were measured in this way.

The two most satisfactory series in which to estimate the cross section of the fasciculus longitudinalis were the two hemispheres of the case of Wilson's disease (figs. 1 and 2). The right one measured 0.373 sq. cm., the left 0.25 sq. cm. A measurement was taken of several levels of the hemisphere from which the wax reconstruction was also made. It contained three lesions, all small; the measurement including them was 0.297 sq. cm. This may perhaps be considered a low normal. The area of the degenerations will be discussed more fully later.

Little of immediate value can be deduced from the size of the fasciculus longitudinalis. A larger series of measurements from normal brains might yield more practical information.

VOLUME OF THE CORPUS GENICULATUM EXTERNUM

The volume of the corpus geniculatum externum was estimated by taking sections at definite intervals—say, every eighth or tenth—drawing it enlarged thirty times by means of a projection camera, weighing the area drawn, calculating the area of each section from the weight, and multiplying the sum of all of them by the distance between the sample

Fig. 6.—Camera lucida drawing of wax plate reconstruction of the central visual system of a baby at term. Right hemisphere, viewed from the mesial side. The corpus geniculatum externum (*C. g. e.*) is heavily shaded. The fasciculus longitudinalis inferior (*F. l. i.*) is lightly shaded; the direction of its fibers is not indicated, but can in general be inferred from the shape of the model. The area in which fasciculocortical fibers lie is shown without shading or stippling. They are diffusely scattered through the region outlined. The striate cortex is deeply shaded. Most of it lies in the depths of the calcarine fissure (*Fiss. calc.*). Present enlargement, × 4.

sections taken. Slightly higher values are obtained from cell-stained sections than from those stained by Weigert-Pal, and the method is at best only approximate.

In the normal frontal series, the volume of the corpus geniculatum externum was estimated at 0.115 cc. In the normal left hemisphere in

case 1 (previously reported [3]), it measured 0.077 cc. In the case from which the wax reconstruction was made, it measured 0.084 cc., including the degeenrated areas. From these figures, a rough idea of the volume of the nucleus may be gained, for comparison with pathologic areas.

This is perhaps the place to call attention to the great variations in the shape of the corpus geniculatum. Typical configurations are illustrated in virtual reconstructions in the preceding paper of this series. The illustrations in Rönne's article [4] also show the range of variation.

The area of cross-section of the optic nerve is 0.07336 sq. cm., and it contains about 550,000 fibers.[5] Thus it is far smaller than the optic radiation.

MYELOGENETIC STUDIES OF THE CENTRAL VISUAL SYSTEM

The conclusions reached in Pfeifer's recent monograph [6] are at variance with some of the results of the present studies. His work, published from Flechsig's laboratory, includes an excellent review of the literature (though many important cases remain unanalyzed) and a large number of very beautiful illustrations, taken chiefly from the brains of children of different ages. For three-dimensional representations, he depends on a peculiar and ingenious method. He had constructed a model of the gray matter of the brain—cortex and basal nuclei—with a space left for the white matter. He then molded various conceivable forms of the optic radiation free-hand, and fitted them successively into the spaces of the concave model. The form which seemed to him to fit the best was that in which fibers from the lower edge of the radiation reached the anterior end of the calcarine fissure, those from the upper edge, the occipital pole. He supposes that macular fibers run in the dorsal portion of the radiation.

In order to account for the connections of both lips of the calcarine fissure by the flat membranous radiation which such a scheme demands, he suggests that there is a crossing of fibers in the subcortical white matter, so that nerve fibers originally dorsal supply the ventral lip of the calcarine fissure. He does not make it clear exactly how this occurs.

Evidently influenced by Heine's [7] theory that a bilateral representation of the macula is implied by the fact of stereoscopic vision, Pfeifer

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3. Putnam, T. J.: (footnote 1, third reference).

4. Rönne, H.: Die anatomische Projection der Makula in Corpus geniculatum externum, Ztschr. f. d. ges. Neurol. u. Psychiat. **22**:469-484, 1914.

5. Zwanenburg, S.: Quantitatief onderzoek over den bouw van het netvlies, Diss., Amsterdam, 1915, pp. 60.

6. Pfeifer, R. A.: Myelogenetisch-anatomische Untersuchungen über den zentralen Abschnitt der Sehleitung, Monographien a. d. ges. Gebiete d. Neurol. u. Psychiat. **43**:146, 1925.

7. Heine, L.: Sehschärfe und Tiefenwahrnehmung, Arch. f. Ophth. **51**:146-185, 1900.

found fibers which seemed to run from the optic radiation toward the corpus callosum in myelogenetic preparations from an infant's brain. The presence of such fibers is illustrated by only one section, however, and no callosal bundle appears in his models.

From this brief review, it may be seen that Pfeifer's scheme is at variance with facts reported by other observers. Most of these facts have been reviewed in the preceding papers of this series, and need not be repeated here. Though some of his conclusions appear erroneous, his monograph should be read by every one who is interested in the finer anatomy of the central visual system, for the sake of its review of earlier work and of its illustrations.

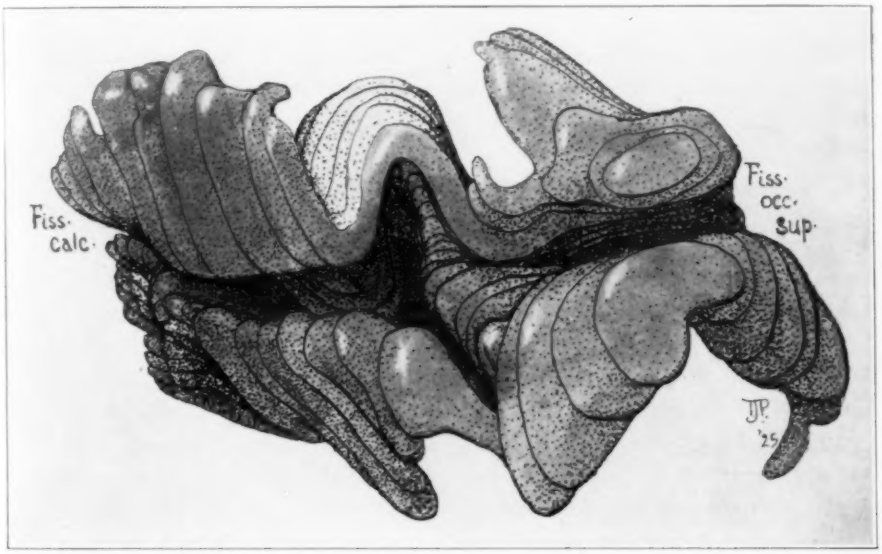

Fig. 7.—Camera lucida drawing of wax-plate reconstruction of area striata alone, disarticulated from the model shown in figure 6; right hemisphere, viewed from the posterior end. The exposed surface of the visual cortex is shaded; the inner surface is shown in white. The calcarine fissure extends far anteriorly on the mesial surface, but much of the area striata lies in the superior occipital fissure (*Fiss. occ. sup.*) laterally. Present enlargement, × 4.

Reconstruction of the Geniculostriate System from a Baby's Brain.—For comparison with Pfeifer's figures, a reconstruction of the geniculostriate system was made from sections of a new-born baby's brain in the possession of Professor Brouwer. Born's wax-plate method was used. This is more laborious than the procedure employed by Pfeifer, but is far more accurate.

There was nothing unusual about the mode of reconstruction itself. Some difficulty was encountered in tracing the precise limits of the fasciculus longitudinalis inferior and of the striate cortex. Toward the occipital pole, where

the relations are complicated, it was necessary to determine the outline of the tract and area striata under the high power of a microscope, to mark it on the slide, and then to project and draw it by means of a drawing camera.

The original model was made with an enlargment of ten. It was cut out in such a way that the geniculate body, fasciculus longitudinalis inferior, fasciculocortical bundles and cortex itself could be separated from one another for study.

The complete model is shown assembled in figure 6. It shows a striking similarity to the reconstruction of an ape's visual system, illustrated in an earlier paper.[8] The "temporal knee" described by

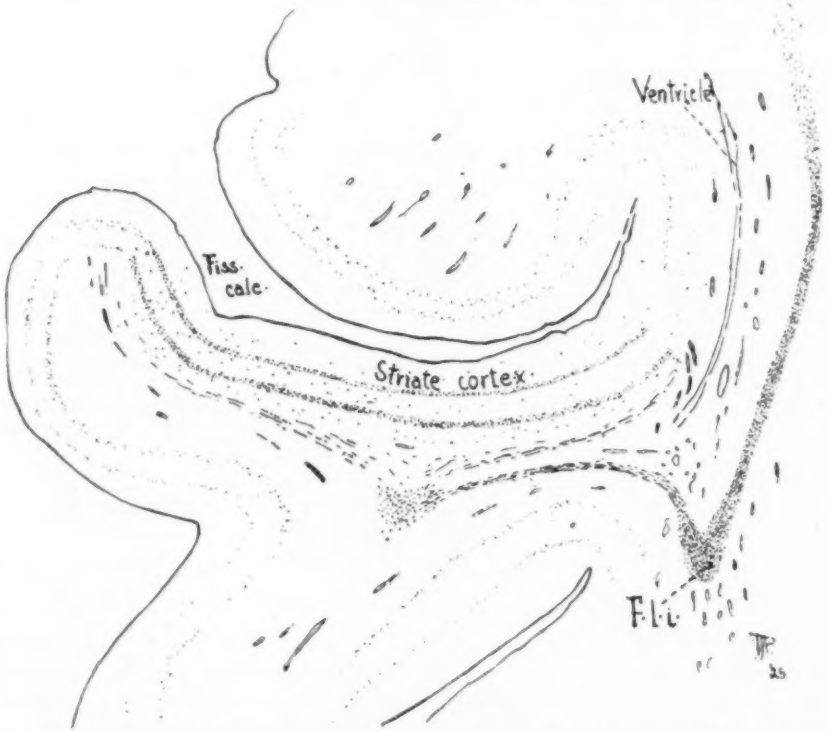

Fig. 8.—Frontal section through the anterior end of the calcarine fissure (*Fiss. calc.*) of the baby's brain. A large subcortical bundle, running vertically to the plane of the section, receives fibers from the inferior edge of the fasciculus longitudinalis inferior (*F. l. i.*) and gives them off to the cortex. A group of large vessels is seen to run to either end of the striate cortex. Weigert-Pal stain; projection drawing; × 50.

Flechsig is obvious anterior to the corpus geniculatum. The form of the geniculate body is somewhat simpler than that seen in adults. The exact curvature of the radiation between nucleus and cortex is difficult to reproduce in the reconstruction as in the illustration. It is best

8. Putnam, T. J.: (footnote 1, second reference).

judged from horizontal sections, such as those given by Pfeifer. It can be seen at once from the reconstruction that the upper lip of the calcarine fissure receives fibers from the tract, not the lower lip alone, as reported by Hösel.[9] Indeed, perhaps a majority of them go to the upper lip. The model also supplies good evidence in itself that the fibers to the upper lip arise from the upper edge of the fasciculus.

A view of the striate cortex alone seen from the mesial and posterior aspect is shown in figure 7. This is reproduced in order to show the extraordinary tortuosity of the depths of the fissure itself, which makes it an uncertain landmark in describing lesions. This was the only brain of those examined during the present study in which a considerable proportion of the striate area lay on the lateral surface of the hemisphere, as in apes.

The sections from this brain show the course of the "bayonet" fibers from compact fasciculus to cortex even more clearly than did those from the ape's brain previously described. It is possible to follow each minute fiber with accuracy. Passing posteriorly, a thin stream of fibers begins to leave the lower end of the fasciculus several millimeters before the area striata is reached. This stream grows coarser. As the striate cortex appears along the lower lip of the calcarine fissure, the scattered fibers running in the plane of the section become gathered into a compact bundle perpendicular to it beneath the middle of the visual area. From this bundle fibers turn at right angles again to run along beneath the cortex, finally plunging into it. This is illustrated by a projection drawing (fig. 8), as photographs of such delicate blue fibers in thick sections are unsatisfactory. Further posterior, a similar fasciculocortical bundle is given off from the dorsal edge of the tract above the ventricle to form a subcortical column above the fissure as the upper lip becomes lined with striate cortex. There is always a thin place in the subcortical white matter where tangential fibers meet from the ventral and dorsal bundles, respectively. No indication of the crossing of fibers described by Pfeifer in this narrow space can be made out. As the configuration of the calcarine fissure becomes more complicated near the occipital pole, more and more bundles of fibers appear beneath the cortex, and others disappear or fuse with them. In the retroventricular white matter, the tract finally frays out entirely into many scattered bundles, some of them supplying the cortex on the lateral surface of the hemisphere.

None of the fibers could be traced into any part of the cortex except the area striata. It is small wonder, however, that their indirect course has led many anatomists to suppose that they were distributed to other

9. Hösel: Ueber die Markreifung der sog. Körpergefühls Sphäre und der Reich- und Sehstrahlung des Menschen, Arch. f. Psychiat. u. Neurol. **39**:195-239, 1905.

portions of the occipital lobe. No fibers are given off from the surface of the fasciculus until the retroventricular space is reached; nor is there evidence that bundles of fibers change their relative positions in the radiation. That is, it appears that the upper and lower edges of the radiation supply the anterior portions of the upper and lower calcarine lips, respectively, and that the central portion of the radiation goes to the posterior portion of the calcarine area.

A peculiar group of coarse vessels could always be seen running to the cortex just adjacent to the area striata. Indeed, these vessels were a useful landmark in locating its extremities under low magnification, where the stripe of Gennari was difficult to see. They are shown at either end of the strip of striate cortex in figure 8. Their significance is not clear. They run to the strip of cortex in which van Valkenburg described a group of coarse fibers lying outside the optic radiation, which he supposed were concerned with movements of the eyes.[10]

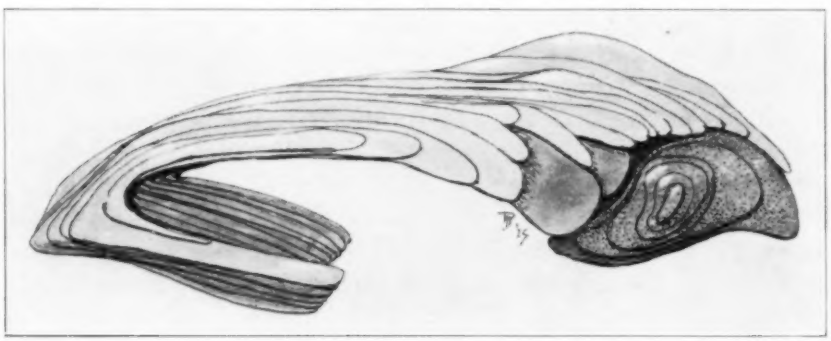

Fig. 9.—Camera lucida drawing of wax-plate reconstruction of model of corpus geniculatum externum (deeply shaded) and "knee" of fasciculus longitudinalis inferior, drawn as if from a right hemisphere, viewed from the anterior aspect. From an adult brain, in which the central visual system was isolated by a lesion in the thalamus (as described in the text). Present enlargement, × 2.

If any callosal fibers were given off from the fasciculus longitudinalis, as Pfeifer believes, they would have appeared in the reconstruction. A careful search was made for them, and they were not found.

Stalk of the Fasciculus Longitudinalis Inferior.—Since the connection between the corpus geniculatum externum and the fasciculus longitudinalis was somewhat confused by fibers which crossed it in various directions, this portion of the radiation was reconstructed from sections of a case previously reported (case 1, left hemisphere [3]), in which the geniculocortical radiation was isolated by a lesion of the

10. Van Valkenburg, C. T.: Experimental and Pathologico-Anatomical Researches on the Corpus Callosum, Brain **36**:119, 1913.

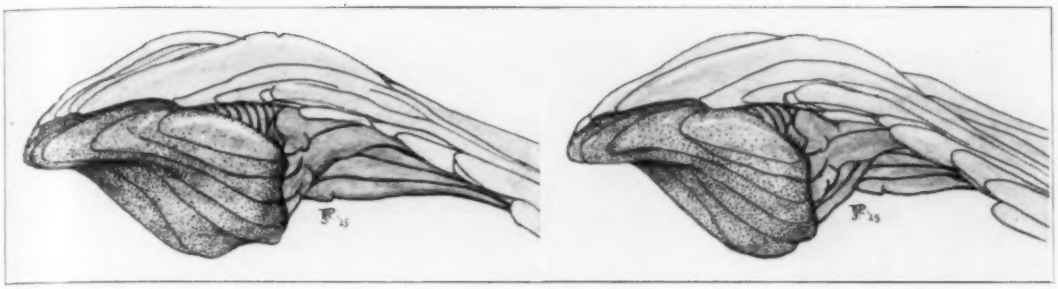

Fig. 10.—Camera lucida drawing from wax-plate reconstruction of stereoscopic appearance of corpus geniculatum externum (deeply shaded) and "stalk" of radiation of the model shown in figure 9. Right hemisphere, viewed from the posterior aspect. The direction of the fibers of the radiation is not indicated, but the model shows that those from the dorsal edge must supply the mesial limb of the nucleus, those from the ventral edge the lateral limb. Present enlargement, × 2.

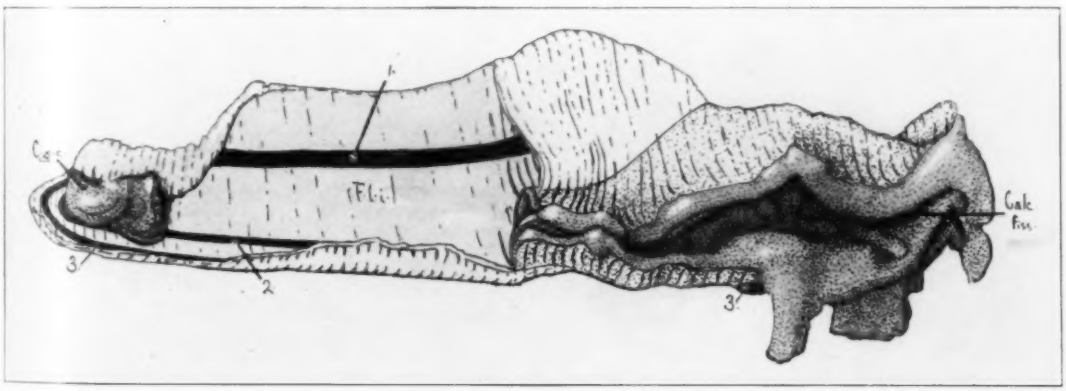

Fig. 11.—Camera lucida drawing from wax-plate reconstruction of model of the central visual system from case 46 (in text), drawn as if it were from a right hemisphere, and viewed from the mesial side. The corpus geniculatum externum (*C. g. e.*) and striate cortex are deeply shaded; the fasciculus longitudinalis inferior (*F. l. i.*) is lightly shaded and the fasciculocortical fibers are left white. The longitudinal degenerations produced by the three vascular lesions are shown in black. A portion of lesion 3 is shown in the lower part of the cortex. Present enlargement, × 1.5.

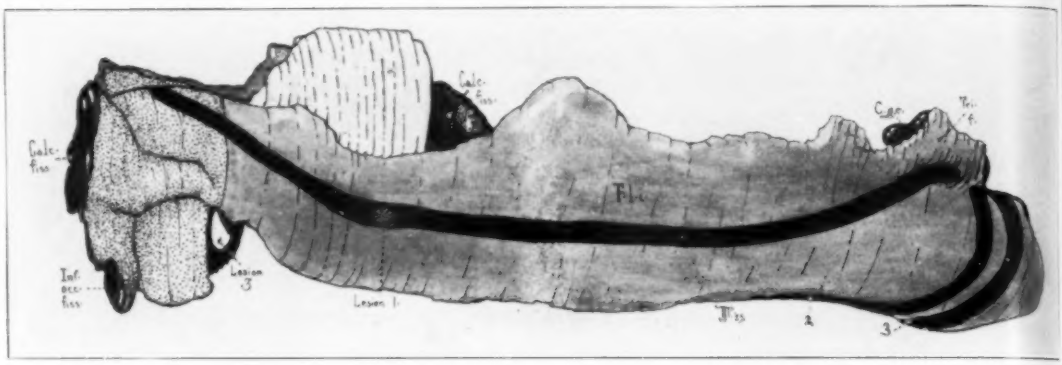

Fig. 12.—Camera lucida drawing from wax-plate reconstruction of model from case 46, seen from the superolateral aspect. Lesion 1, in the fasciculus longitudinalis inferior, and part of lesion 3 in the cortex are both shown. All three degenerations can be traced forward into the triangular field of Wernicke (*Tri. f.*). Posteriorly, the end of the inferior occipital fissure (*Inf. occ. fiss.*) is shown for orientation. The anterior end of the lower lip of the calcarine fissure is seen to extend forward of the upper lip. Present enlargement, × 1.5.

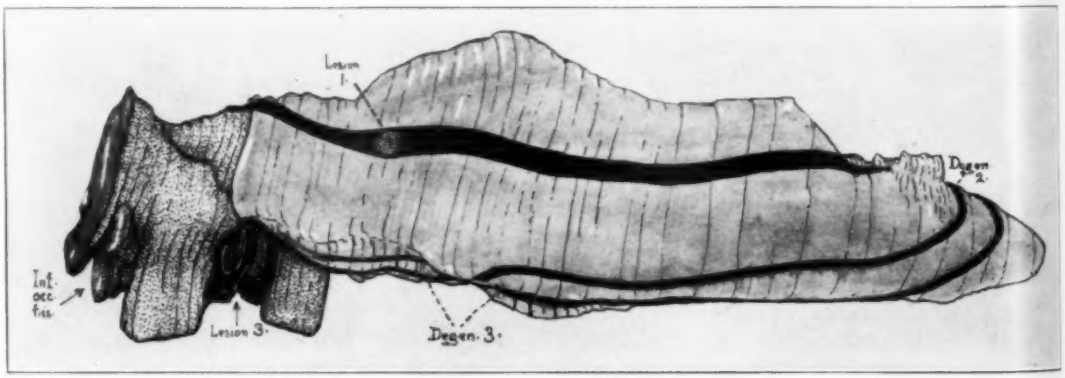

Fig. 13.—Camera lucida drawing of model from case 46, viewed from the inferolateral aspect. Notations are given as in figure 12. Present enlargement, × 1.5.

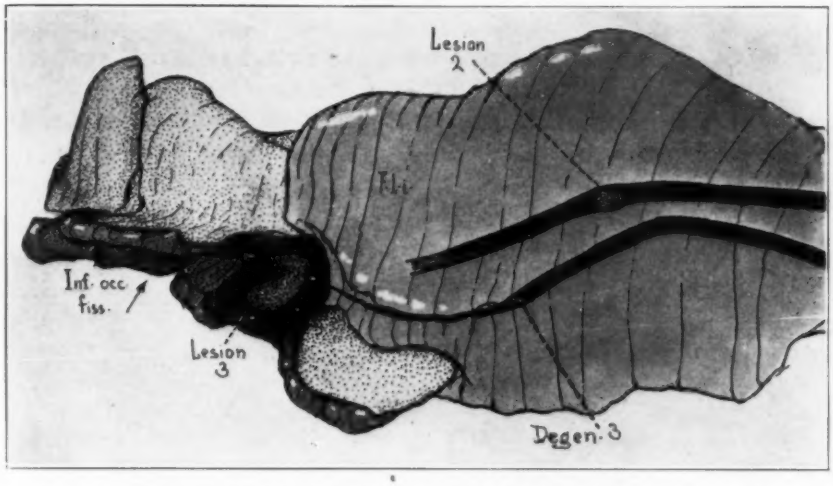

Fig. 14.—Camera lucida drawing of model from case 46; posterior end, viewed from beneath, to show relations of lesions 2 and 3. Present enlargement, × 1.5.

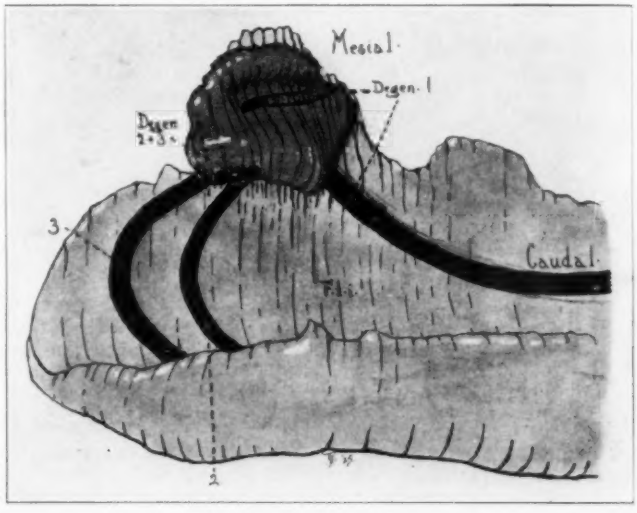

Fig. 15.—Model of case 46; anterior end, viewed from beneath, to show the degenerations in the stalk of the fasciculus longitudinalis inferior (*F. l. i.*) and in the corpus geniculatum externum. Degeneration 1 is shown in black in the corpus geniculatum where it reaches the inferior surface; it is outlined and heavily stippled where it lies in the substance of the nucleus (as described in the text). The degeneration produced in the geniculate body by lesions 2 and 3 is slightly exaggerated. Present enlargement, × 1.5.

thalamus. Figure 9 shows the anterior side of the entire reconstruction, and figure 10 is a stereoscopic drawing showing the corpus geniculatum and "stalk" of the radiation more in detail (viewed from behind). It may be seen that the external form of the fiber bundles entering the nucleus can scarcely be explained except on the basis that the upper edge of the radiation supplies the mesial horn of the corpus geniculatum, the lower edge the lateral horn.

Course of Localized Degenerations in the Fasciculus Longitudinalis.—The study of normal anatomic relations thus lends support to the results of clinical and pathologic study. It does not identify the origin and termination of individual groups of fibers, however. To determine this, a reconstruction was made from sections in case 46 of the Institute for Brain Research, already reported by Brouwer (case 2, left hemi-

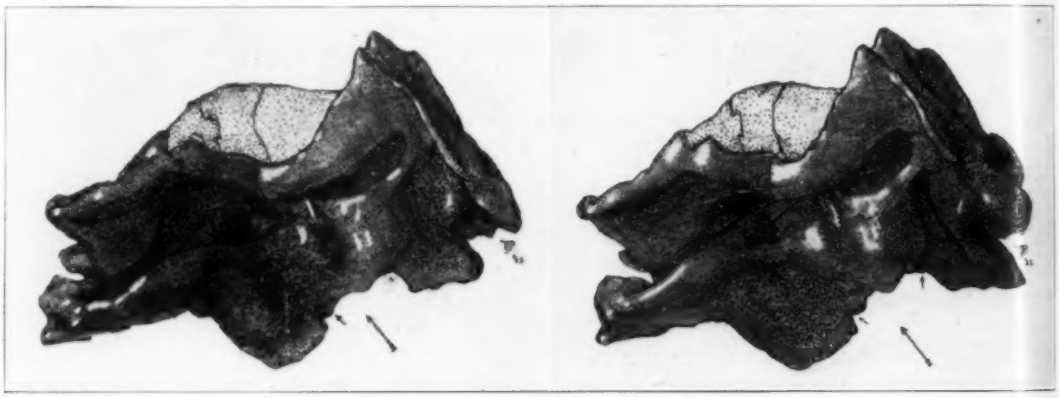

Fig. 16.—Camera lucida drawing of wax-plate reconstruction of stereoscopic appearance of the area striata alone, from the model of case 46, drawn as if from a right hemisphere, viewed from the mesial side. The lips of the fissure are slightly spread apart. The position and extent of lesion 3 is indicated by arrows. About natural size.

sphere [2]), to which many references have already been made. The case is described in the original in detail, with many sketches and photographs of different levels of the brain.

We may here confine our attention to the left geniculocortical system (drawn in the illustrations as if it were from the right hemisphere, for the sake of uniformity). A close study of the sections during the preparation of the reconstruction revealed three discrete lesions. Passing anteriorly from the occipital pole, the first one encountered (lesion 3 of the illustrations) lies in the cortex, and does not involve the radiation directly. An area of cortex measuring 1.64 sq. cm. has been destroyed. It is impossible to follow individual fasciculocortical fibers, but the

diffuse degeneration of the white matter can readily be traced into the fasciculus longitudinalis inferior, where it becomes more localized as a small pale area near the angle between the horizontal and the vertical limb.

The next lesion is a small hemorrhagic cyst in the upper part of the fasciculus, a little posterior to the ventricle. It can be followed in both

Fig. 17.—Angle of the fasciculus longitudinalis inferior (outlined by arrows) from case 46. The degeneration due to lesion 2 is seen above. It consists of a narrow band of practically complete degeneration, running obliquely across the fasciculus. Lesion 3 (below) is more diffuse, and a few well-preserved, coarse fibers appear in it. Weigert-Pal stain; ×200.

directions, posteriorly to the point at which the radiation is last seen as a distinct bundle, anteriorly into the corpus geniculatum. The most anterior lesion (lesion 2) also involves the radiation alone. It is smaller than lesion 1 and cannot be followed so far posteriorly. Anteriorly, the

tiny streak of degeneration holds its place just above that caused by lesion 3 (fig. 17).

These lesions and the degenerations caused by them were carefully traced on successive plates of the wax model, and lines were painted on the surface of the finished reconstruction to show their course. All of the degenerations ran horizontally, in the long axis of the tract. The intervals between the respective degenerations remained approximately the same from cortex to nucleus. Degeneration 1 could be traced from its origin to the lesion in the corpus geniculatum, illustrated in Brouwer's article,[11] a small streak of atrophy, perpendicular to the laminae of the nucleus, slightly mesial to its middle point posteriorly. The atrophy gradually becomes confined to the upper layers of cells anteriorly, and is not seen in the anterior third of the nucleus. Degenerations 2 and 3 become confused as they approach the geniculate body, but they appear to terminate in an inconspicuous area of atrophy in the tip of its lateral horn (fig. 15).

A stereoscopic drawing of the cortex is reproduced (fig. 16) to show more exactly the relationships of lesion 3. It also serves to give an idea of the complexity of the surface of the striate cortex in the adult.

COURSE OF DEGENERATIONS DUE TO LESIONS IN VARIOUS SITUATIONS

Study in case 46 by means of a reconstruction confirms the "vertical articulation" of the geniculocortical radiation, which we have already seen to be supported by other facts. Consideration of the sections in series also reveals an important difference between the degenerations due to lesions of the fasciculus longitudinalis inferior, and those due to lesions of the cortex. The former vary little throughout their course, though becoming somewhat encroached on by neighboring fibers; the latter are almost from the first diffuse and incomplete, as will be seen by comparing lesions 2 and 3, in figure 17. Different bundles of fibers must therefore keep their relative positions accurately in the radiation, as was pointed out by Meyer,[12] but adjacent fibers in the radiation do not necessarily supply closely adjacent points in the cortex. That there is some shifting of fibers in the fasciculus is shown also by the fact that perimetry often discloses a great incongruence between the defects in either visual field, in cases in which there is an injury to the anterior portion of the radiation,[13] while scotomas due to posterior lesions of the radiation or cortex are usually superposable. This is the opinion

11. Brouwer, B.: Ueber die Sehstrahlung des Menschen, Monatschr. f. Psychiat. u. Neurol. **41**:155, 1917.

12. Meyer, A.: The Connections of the Occipital Lobes and the Present Status of Cerebral Visual Affections, Trans. A. Am. Phys., 1907, pp. 1-10.

13. Cushing, Harvey: Distortions of the Visual Fields in Cases of Brain Tumor, Brain **44**:341-396 (Jan.) 1922.

reached by Minkowski [14] by a different path. In case 46, degenerations 2 and 3 both appear to be displaced somewhat laterally as if by the accession of new fibers along the mesial side, as the series is followed forward.

Representation of the Macula in the Geniculostriate Radiation.—In the corpus geniculatum, the degeneration due to lesion 1 lies almost but not wholly within the area allotted to the macula by Rönne [4] and by Brouwer and Zeeman.[15] Posteriorly, its lower end appears to extend somewhat lateral of the macular field. In the radiation, therefore, lesion 1 must approximately represent the upper boundary of the macula region, lying between the upper and the lower quadrants. In Brouwer's case 1, in which there were lesions of the tips of both occipital lobes involving also the central portion of the fasciculus longitudinalis, the edges of the radiation were preserved. In the corpus geniculatum, the degenerations lay chiefly within Rönne's macular area on both sides.

This situation of the macula is suggested by many clinical observations. Holmes [16] was led to predicate the existence of an anatomic interval between the two retinal quadrants by the enormous predominance of scotomas bounded by the horizontal line among partial field defects due to war wounds, and Rönne [17] suggests further that this interval is occupied by the macular bundle. He points out that there is a developmental basis for such an arrangement. Fibers from the periphery of the retina run radially to the nerve head in the nasal field, but the temporal field is divided into two quadrants by a horizontal line, from which fibers swing in an arc around the macula. Ariëns Kappers [18] has found evidence to show that in the regrouping of optic fibers for binocular vision, the nasal elements have adopted the pattern of the temporal elements, as the latter were first employed simultaneously. Two cases reported at different times by Lenz,[19] in which lesions of the

14. Minkowski, M.: Ueber den Verlauf, die Endigung, und die zentralen Repraesentation von gekreuzten und ungekreuzten Sehnervenfasern bei einigen Säugetieren und beim Menschen, Schweiz. Arch. f. Neurol. u. Psychiat. **6**:201-252 and 268-303, 1920.

15. Brouwer, B., and Zeeman, W. P. C.: Experimental Anatomical Investigations Concerning the Projection of the Retina on the Primary Optic Centers in Apes, J. Neurol. & Psychopath. **6**:1-10 (May) 1925.

16. Holmes, G.: The Cortical Localization of Vision (Montgomery Lecture), Brit. M. J. **2**:193-199 (Aug. 16) 1919.

17. Rönne, H.: Ueber Quadranthemianopsie und die Lage der Makulafasern in der okzipitalen Sehbahn, Klin. Monatschr. f. Augenh. **63**:358-374, 1914.

18. Kappers, C. U. A.: Die vergleichende Anatomie des Nervensystems der Wirbeltiere und des Menschen, part 2, Harlem, 1921, pp. 898-900.

19. Lenz, G.: Zur Pathologie der cerebralen Sehbahn unter besonderer Berücksichtigung ihrer Ergebnisse für die Anatomie und Physiologie, Arch. f. Ophth. **72**:1-85 and 197-273, 1909; Zwei Sektions von Fälle doppelseitiger zentraler Farbenhemianopsie, Ztschr. f. d. ges. Neurol. u. Psychiat. **73**:135-186, 1921.

upper edge of the radiation produced lower quadrant peripheral scotomas, are additional support for this relationship.

There is also reason to believe that the macular field overlaps the regions devoted to the upper and lower quadrants in the geniculostriate radiation. Lesion 1 of the present case is an almost perfect transverse division of the fasciculus, and yet apparently involves some fibers coming from the area in the geniculate body supplied by the upper quadrant of the retina. In the other hemisphere of the same brain, in which

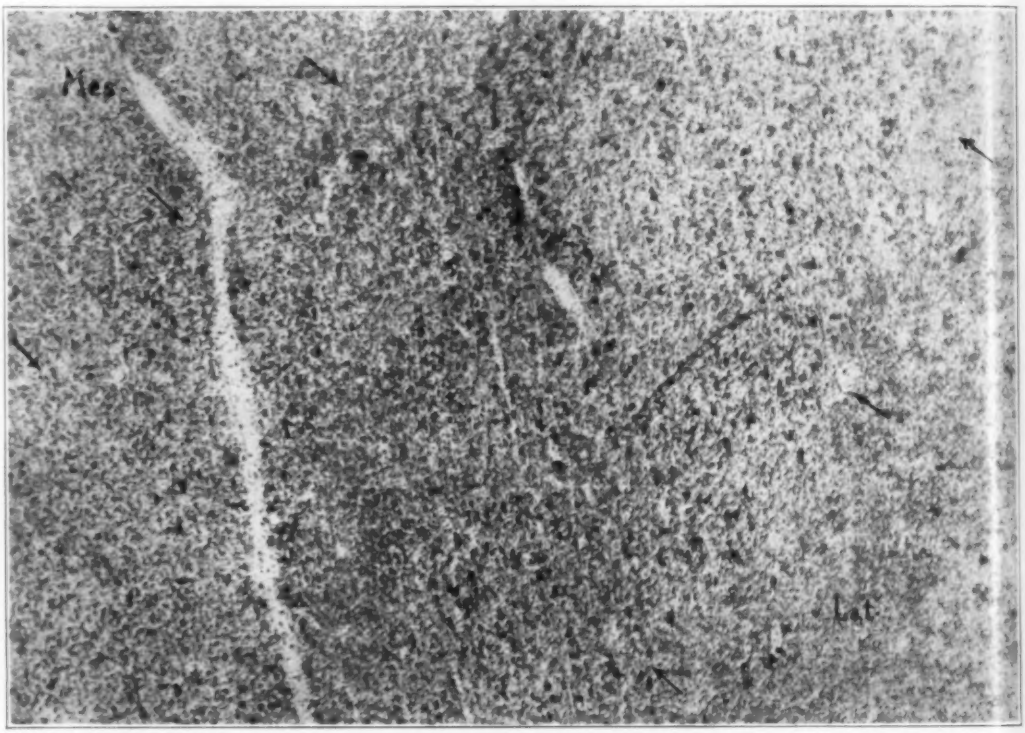

Fig. 18 (case 1 [Holman] of the preceding paper).—Middle portion of fasciculus longitudinalis inferior (indicated by arrows). The surviving striate cortex lies at the tip of the occipital pole. The surviving visual projection fibers (probably mostly macular) lie scattered through the thickness of the fasciculus. Their coarse caliber is well shown. Weigert-Pal stain; × 400.

all that is left of the area striata is a small bit measuring 1.63 sq. cm. anteriorly, mainly in the upper lip of the fissure, scattered coarse fibers are found not only at the upper end of the fasciculus longitudinalis, but also in small numbers almost down to its center. This may be compared with Brouwer's article, plate III, figure 11. The right hemisphere of the first case reported in the preceding paper of this series illustrates

the same point. The only surviving visual cortex lay at the occipital pole; the preserved coarse fibers lay scattered throughout the thickness of the radiation, a few extending even to its edges (fig. 18).

An overlapping of the macular area with the areas devoted to the retinal quadrants is to be expected by analogy with the optic tract, where in cross section the macular fibers form a triangle with the base mesial.

The fact that lesions 2 and 3 are displaced somewhat laterally, apparently by the acquisition of new fibers from the anterior portion of the visual cortex, suggests that the tip of the occipital lobe is represented along the external surface of the central part of the fasciculus longitudinalis inferior. But it is not justifiable to draw too fine conclusions on this point, as figure 18 shows the persisting fibers from a preserved area at the posterior extremity of the area striata to be scattered diffusely throughout the thickness of the radiation.

Proportion of the Optic Nerve Devoted to Macular Fibers.—Zwanenburg [5] made an estimation of the number of cells of different kinds in various zones of the retina, and of the number of fibers in the optic nerve. The complexity of the nervous connections within the retina is too great to be considered here, but it appears that the ganglion cells are the origin of the third neuron in the peripheral chain, and that the fibers of the optic nerve are derived from them. Zwanenburg found 29,000,000 ganglion cells in the central territory with a radius of 2.7 mm. about the fovea (corresponding to about 25 degrees), and only 16,500,000 in all the rest of the retinal surface. That is, the central territory apparently gives rise to about 65 per cent of all the optic fibers. It is questionable whether this central territory, or a much smaller one comprising only the concavity about the fovea, represents the macula as the term is used in perimetry. The comparison in the latter case between the macular and the peripheral fields would then be even more striking

Macular Representation in the Corpus Geniculatum.—Unfortunately, no data are at hand on which to base even a rough estimate of the proportion of cells in the corpus geniculatum devoted to macular vision. Neither the results of Brouwer and Zeeman nor those of Rönne were mathematically quantitative. At most we may gain the impression from their illustrations that the central part of the retina is no less well represented in the geniculate body than in the optic tract.

Area of the Macular Representation in the Geniculostriate Radiation.—A crude idea of the size of the macular area in the fasciculus longitudinalis inferior may be gained from measurements in case 46. The accuracy of the estimation is limited by the correctness of the assumption that lesion 1 lies almost wholly within the macular area, and by the correctness of the measurements possible. Accepting the two large sources of error, we may proceed as follows: The total area of the cross-section of the fasciculus is 0.297 sq. cm. (average of five esti-

mations). The area of the segment above lesion 1, which we are assuming to represent the upper retinal quadrant, is 0.08 sq. cm. If the lower retinal quadrant occupies a similar area, the remainder—0.137 sq. cm. or about 47 per cent—must be devoted to macular fibers. This figure is doubtless too low, as any shrinkage of the radiation due to atrophy would be subtracted from the macular area.

A similar calculation might be made, for example, in a case given by Lenz,[19] in which a loss of about 2 mm. of the upper edge of the radiation, of which the total width was 28 mm., caused a lower quadrantic

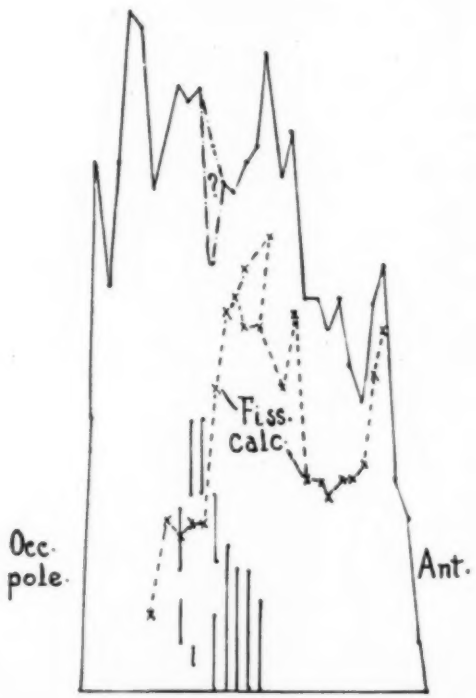

Fig. 19 (case 46).—Plane map of area striata. The space occupied by the lesion (about the middle of the base line) is shown by vertical lines. The total area is about 24.57 sq. cm. and that of the lesion alone, 1.64 sq. cm. Actual size.

total peripheral scotoma to within 30 degrees of the fixation point. But we must not throw too much burden on the few facts available.

Shape and Area of the Macular Representation in the Cortex.—Is the line dividing the macular from the peripheral territory in the cortex vertical, or does the macular field fit between the upper and lower fields like a wedge, as in the corpus geniculatum and optic tract? It must be admitted that the evidence is conflicting. On the one hand, there is the important case of Holmes,[15] in which a concentric defect larger than the

macula was caused by a lesion destroying all the posterior 3 cm. of the occipital lobe. But without the fixation point, perimetry is uncertain, and the case was not studied microscopically.

More exact evidence seems to be furnished by case 46, and one of those from the previous paper. In case 46, lesion 3 has produced a degeneration which lies outside the macular area in the fasciculus longitudinalis. We cannot say that no macular fibers pass through it, but we should not expect it to be included in the macular area of the cortex. Figure 19 shows its location, at the lower edge of the striate area, within 24 mm. of the occipital pole. On the other hand, the macular territory cannot extend much farther forward than 30 mm. from the pole, at which point lesion 1 occurs, causing complete atrophy of a bundle from it. In case 1 (Holman) of the previous paper, the atrophy in the corpus geniculatum extended into both mesial and temporal horns over a small area posteriorly; hence a few fibers from both the upper and the lower retinal quadrants must have survived; yet the anterior portion of the striate cortex was entirely destroyed to within 10 mm. of the

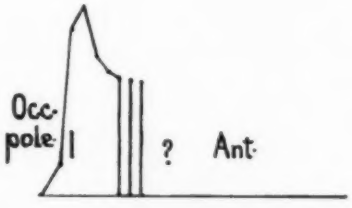

Fig. 20 (case 1 [Holman] of preceding paper).—Plane map of persisting area striata, at tip of occipital pole. The normal portion measures 1.38 sq. cm. Actual size.

occipital pole (fig. 20). It seems likely, then, that the "cortical macula" is triangular in outline, with its apex anterior.

A rough estimation of the macular territory in case 46 may perhaps be made by erecting an equilateral triangle with its base toward the occipital pole within the outline of figure 19, in such a way as to avoid lesion 2. The minimum area thus outlined must contain at least 390 sq. mm. This figure is probably no more than half the correct one, judging from the proportion of fibers in the radiation that are devoted to the macula.

SIGNIFICANCE OF THE PROPORTIONAL REPRESENTATION OF THE MACULA IN THE VISUAL SYSTEM

In the first paper of this series,[1] it was shown that fibers serving binocular vision in the rabbit are scanty in the optic nerve, but that a larger area in proportion is allotted to them in the corpus geniculatum, and perhaps a still larger proportionate area in the cortex. This might

almost be predicated. In a cortical area devoted to binocular vision, not only must there be an element representing each retina for each visual unit, but also a mechanism for blending their separate impressions and for transmitting them to centers for stereoscopic perception.[20] Thus each visual unit requires at least twice as much space in the corpus geniculatum, and at least three times as much space in the cortex for binocular perception as for monocular perception.[21] Once given binocular vision, macular impressions require no more elaboration than do peripheral ones, only there are more of them per unit of visual angle. Thus, the proportion of nerve, of corpus geniculatum, of radiation and of cortex devoted to macular fibers remains approximately constant.

It should be emphasized again that the clinician's conception of the macula is not a perfectly definite one, as is that of the binocular field. Sometimes the macular field is taken to cover 5 degrees, sometimes 25. It is particularly variable in size in the papers of those who support the doctrine that it is represented bilaterally. Until more unanimity is reached as to what is to be considered the size of the macula in the perimetric field and in the retina, one cannot expect to be definite as to the shape and size of its representation elsewhere in the visual system.

LIMITATIONS OF THE PRESENT PAPER AND QUESTIONS REMAINING UNANSWERED

There is sufficient correspondence between the three models described in this paper and those of lower animals previously reported, to entitle them to some degree of confidence. They agree in many particulars with Meyer's glass model;[12] in regard to the distribution of fibers to the occipital cortex, new facts have since been disclosed which make his scheme seem less probable than the one suggested here. The anatomic observations speak for themselves.

In the interpretation of the physiologic significance of different parts of the central visual system—the location of the macular area in cortex and radiation, for instance—we are on less certain grounds. The evidence at hand is fairly consistent, but more of it is urgently needed. It is, for example, deplorable that no case of hemianopia with preservation of central vision has been reported in adequate detail, with satisfactory fields and intelligible measurements of the remains of the striate

20. Kleist, K.: Die einzeläugigen Gesichtsfelder und ihre Vertretung in den beiden Lagen der verdoppelten inneren Körnerschicht der Sehrinde, Klin. Wchnschr. **5**:3-10 (Jan. 1) 1926.

21. In the human brain, the area reserved for monocular vision has never been demonstrated anatomically; it is probably so small that it may be neglected.

cortex, radiation and corpus geniculatum. The quantitative results here presented are still less certain, resting as they do largely on the measurements of a single case. Perhaps the best that can be said for them is that they are better than none, and that they have served to develop a method which may prove useful in the future.

There is, then, a need for corroboration of these results. In addition, there remain many interesting questions which the method of study here employed does not answer. Thus, no satisfactory cases of a localized partial lesion of the retina, nerve tract or corpus geniculatum have been published. We have almost no positive information as to the function of the internal sagittal stratum. No satisfactory explanation has been given of the presence of the ventral rows of very large cells in the corpus geniculatum and their persistence in the midst of a profound atrophy of the upper layers. The fasciculus longitudinalis inferior offers an excellent opportunity for investigation into the neurobiotactic (or hodologic) influences which cause the peculiar "bayonet" course of its fibers. There is, in short, still a wealth of opportunity for investigation in connection with the central optic pathways.

CONCLUSIONS

1. In a study of the central visual system in three dimensions and quantitative estimations of the size of its component parts, and of lesions of them, the proportionate area devoted to central vision appears to be approximately the same in optic nerve tract, corpus geniculatum, inferior longitudinal fasciculus and striate cortex.

2. The macula appears to be represented in a wedge-shaped area, with its apex anterior, in the striate cortex. This area extends not less than 2 and not more than 3 cm. anterior to the occipital pole in the specimen studied.

3. In the fasciculus longitudinalis inferior, the macular fibers lie chiefly in the central third, but overlap the upper and lower quadrant fibers above and below, and mingle with them at the edges of the radiation.

4. Projection fibers proceeding from homologous points in the two retinas are farthest apart at the corpus geniculatum, and gradually approach one another as they draw near the cortex.

5. Further evidence is given of the "bayonet" course of fibers between the radiation and the cortex.

Without the most friendly assistance of Dr. Ariëns Kappers, Director of the Central Institute for Brain Research, and of Professor Brouwer of the University of Amsterdam, these observations would have led to scanty results. I am glad of an opportunity to express my gratitude to them both.

A CASE OF AMAUROTIC FAMILY IDIOCY

LATE INFANTILE TYPE (BIELSCHOWSKY) WITH CLINICAL PICTURE OF DECEREBRATE RIGIDITY *

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In contrast to the great number of clinical and pathologic reports on the infantile (Tay-Sachs) type of amaurotic family idiocy, reports on the juvenile and especially the so-called late infantile (Bielschowsky) types are scarce. This is most likely due to the fact that the last two varieties are rare and do not present such a clear cut clinical picture as the classic form of Tay-Sachs disease. For this reason also they are usually not recognized by clinicians but are accidental observations postmortem.

Described first by Jansky,[1] cases of the late infantile variety of amaurotic family idiocy have been recorded by Schob,[2] Brodmann [3] and Marinesco,[4] and have been especially well studied by Bielschowsky.[5] The case here recorded is remarkable not only for its pathologic but also for its clinical features, which were those of decerebrate rigidity, and for the fact that the disease occurred in one of twins.

REPORT OF CASE

A Hungarian-American girl, Roman Catholic, aged 7 years and 3 months, was admitted, Aug. 19, 1925, to the pediatric service of the Cook County Hospital because of inability to talk, walk or take nourishment. The father stated that the

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* From the Division of Neurology of the University of Illinois College of Medicine, and the Pathologic Laboratories of the State Psychopathic Institute and the Research and Educational Hospitals of the University of Illinois.

1. Jansky, J.: Ueber einen noch nicht beschriebenen Fall der familiären amaurotischen Idiotie mit Hypoplasie des Kleinhirns, abstr., Ztschr. f. d. Erforsch. u. Behandl. d. jugendl. Schwachsinns **3**:86, 1910.

2. Schob, F.: Zur pathologischen Anatomie der juvenilen Form der amaurotischen Idiotie, Ztschr. f. d. ges. Neurol. u. Psychiat. **10**:303, 1912.

3. Brodmann, K.: Fall familiärer Idiotie mit neuartigem anatomisch. Befund, Ztschr. f. d. ges. Neurol. u. Psychiat. (Referate) **10**:91, 1914.

4. Marinesco, G.: Contribution à l'étude anatomo-clinique et à la pathogenie de la forme tardive de l'idiotie amaurotique infantile, J. f. Psychol. u. Neurol. **31**:210, 1925.

5. Bielschowsky, M.: Ueber spät-infantile familiäre amaurotische Idiotie mit Kleinhirnsymptomen, Deutsche Ztschr. f. Nervenh. **50**:7, 1914; Zur Histopathologie und Pathogense der amaurotischen Idiotie mit besonderer Berücksichtigung der zerebellaren Veränderungen J. f. Psychol. u. Neurol. **26**:123, 1920.

patient, one of twins, was born at full term by normal delivery, and weighed 4½ pounds (2 Kg.). The child was breast fed, was able to sit up at the age of 6 months, cut her first teeth at 10 months, walked at 13 months, and talked at the age of 2 years. When she was 3 years old she sustained a head injury after a fall which rendered her cyanotic and from which she apparently recovered fully. Six months after the injury, at the age of 3½ years, the child began to show speech troubles and difficulty in walking. During the following four years, up to the time of her admission to the hospital, the inability to walk and talk grew steadily worse, she was losing weight and became untidy, and marked clonic contractions appeared in the face, eyelids and extremities.

The other twin, a girl, is living and well. Venereal diseases were denied by the mother.

Examination.—The child was anemic and lay motionless on her back, the head in the median line and retracted, the eyes and mouth half open, the arms semiflexed at the elbows, and the hands and fingers markedly flexed; the legs were adducted and extended in the hip and knee joints; the feet were in plantar flexion, in a position of equinovarus. There was total absence of voluntary movements; the child was unable to chew, talk or swallow. The eyelids and the muscles of the lower extremities exhibited constant twitchings, while the muscles of the neck, the extensors of the legs and the flexors of the arms were markedly rigid. On rotation of the head, with the chin toward the left shoulder, the left forearm after a few seconds became slowly extended at the elbow, and the leg on the corresponding side becoming more rigid, while the opposite extremities became more flexed at the elbow and at the knee. A similar procedure—rotation of the head to the right—caused extension of the right forearm and leg, with flexion of the contralateral extremities. Ventral flexion of the head was not accompanied by changes in the position of the extremities. Symmetrical labyrinthine reflexes were not tested.

The tendon reflexes were all exaggerated; the abdominal reflexes and the Babinski and similar signs were absent; the pupils were equal, regular and semidilated, and reacted sluggishly to light. Strabismus, nystagmus and facial asymmetry were absent, as were also the Kernig and Brudzinsky signs. Pinpricks or other stimulations elicited no or very feeble responses.

Ophthalmoscopic examination (Dr. Suker) revealed bilateral optic atrophy, "probably postneuritic, secondary to papilledema, with visible choroid vessels. Four or five tuberculous nodules were seen on the choroid of the right retina. The impression is that we are dealing with a tuberculoma."

Spinal puncture yielded a clear fluid, with 8 cells per cubic millimeter; the Pandy test was negative and the pressure was not increased. Blood examination: hemoglobin, 60 per cent; red cells, 2,800,000; white cells, 12,600, of which 78 per cent were polymorphonuclear, 22 per cent lymphocytes. The Wassermann test with the spinal fluid and the tuberculin test were negative. A roentgen-ray examination of the sella turcica gave negative interpretations.

Course.—An abstract of the notes made by the resident physician (Dr. Lifvendahl), Aug. 29, 1925, is: "The Magnus and the de Kleijn phenomenon is only partially elicited; the extensor muscle hypertonus in all four extremities during the latter procedure is of a partially plastic type which seems to be more marked than heretofore; the tendon reflexes show a prolongation, while the spinal reflexes offset the rigidity."

The temperature ranged from 99.8 to 101.4 F.; the pulse rate ranged from 78 to 110; the respirations from 24 to 28. The clonic muscular twitchings, marked at the time of admission to the hospital, became, on September 4, practi-

cally continuous. On September 9, the child became stuporous, but the twitchings did not cease. Artificial feeding, nasal and rectal, was necessary. Death occurred four days later with signs of bronchopneumonia.

Clinical Diagnosis.—As the optic atrophy was interpreted as secondary to papilledema, a diagnosis of a brain tumor, probably in the region of the sella turcica, was suggested.

Anatomic Diagnosis.—The necropsy, performed by Dr. C. C. Guy, resident pathologist of Cook County Hospital, revealed: diffuse "scarring" of the leptomeninges; external and internal hydrocephalus; marked generalized pressure atrophy of the brain; flexion deformities of the upper and extension deformities of the lower extremities; generalized emaciation; bilateral bronchopneumonia; bilateral hypostatic pulmonary hyperemia and edema; fibrocaseous tuberculosis of the tracheobronchial lymph glands; right focal fibrous pleuritis; fatty changes of the liver; cloudy swelling of the parenchymatous organs, multiple decubitus ulcers over the sacrum; fecal distention of the sigmoid and rectum.

MACROSCOPIC OBSERVATIONS

The convolutions of the brain were markedly atrophied, especially in the frontoparietal area; the sulci were gaping and the pia was opaque, fibrous and thickened, especially over the right frontal lobe, but it was practically normal over the cerebellum and was nowhere adherent; the blood vessels were markedly hyperemic; the ventricles were greatly dilated and the ependymal layer was smooth; no evidences of tumor, softening or hemorrhage were found; the hemispheres and the middle lobe (upper and lower vermis) of the cerebellum were greatly reduced in size (fig. 1) and like the cerebral hemispheres were hard and leathery to the touch; the lamellae appeared atrophied and loosely attached to each other; in short, the whole cerebellum was markedly atrophied.

MICROSCOPIC EXAMINATION

The ganglion cells of the cortex formed the usual layers, except in the occipital lobe (fig. 2). Here the third, fourth and fifth layers were practically destroyed and replaced, especially the third layer (a white, colorless stripe), by fibrous neuroglia tissue. In general, the ganglion cells throughout the brain, cerebellum, pons, medulla and spinal cord showed changes that strikingly resembled those of the classic form of amaurotic family idiocy of Tay-Sachs. The cell bodies in their basal portion were spherical, pear shaped (fig. 3) and swollen; the Nissl substance was scanty and gathered around the nucleus which was rich in chromatin and pushed toward the apical dendron; the dendrons were for the most part reduced in number and in some cells totally lacking; in rare instances, they were swollen, containing, like the expanded basal portions of the cell bodies, a granular substance (prelipoids); the neurofibrils were displaced to the periphery, the basal portion of the body appearing reticular and exhibiting a distinct so-called endocellular plasmatic network. The latter was especially in evidence in the ganglion cells of the deeper strata of the cerebral cortex, the bulbar nuclei and the anterior horn cells of the spinal cord (fig. 4). In the latter the ganglion cells appeared much better preserved than in any other region of the brain and in many instances appeared quite normal.

Another group of ganglion cells was practically devoid of Nissl bodies. Their swollen bodies were reticular, represented by an endocellular network; the fibrils making up the latter were exceedingly delicate and thin, often broken up and transformed into dustlike matter. The nucleus in such cells was often reduced

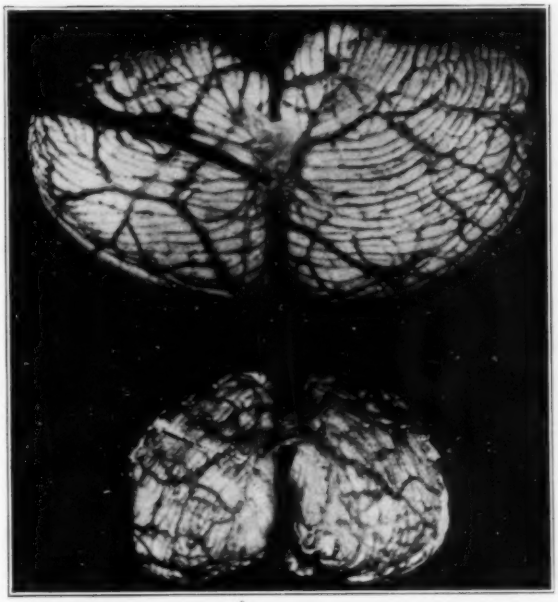

Fig. 1.—The lower picture represents the atrophied cerebellum; the upper, a cerebellum from a patient of the same age.

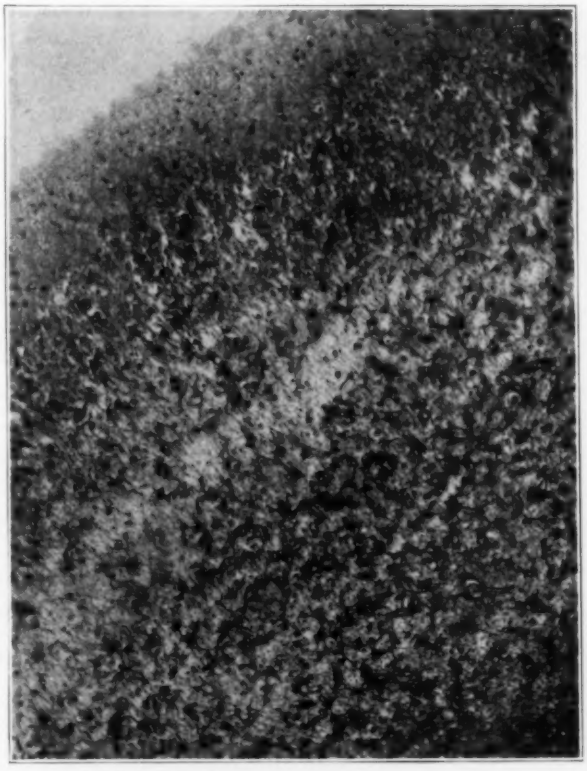

Fig. 2.—Occipital lobe-calcarine fissure: the lower strata, especially the third layer (white stripe), are replaced by a markedly proliferated glia tissue. Holzer's glia stain; × 80.

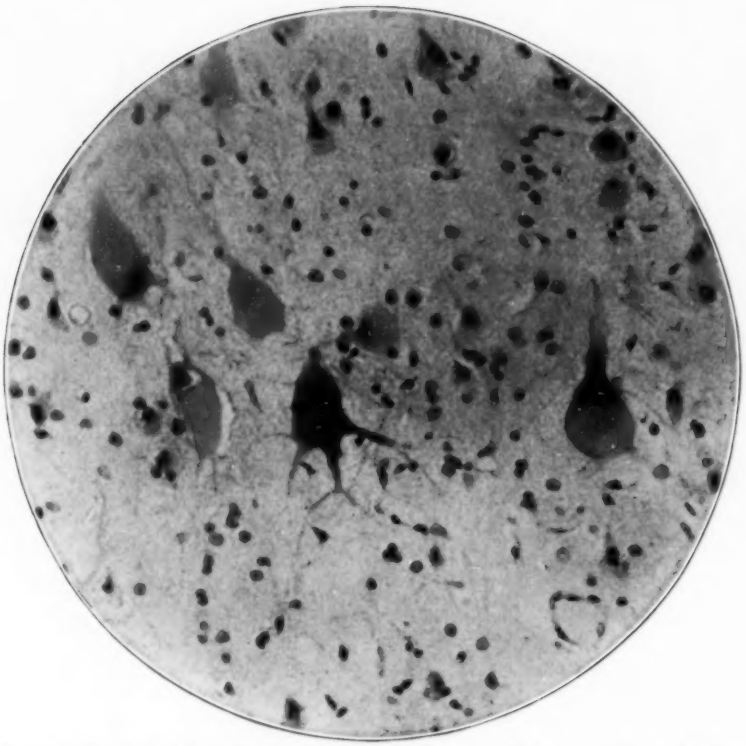

Fig. 3.—Frontal lobe: the ganglion cells are swollen, pear shaped; the basal portion is devoid of Nissl substance and the nucleus is misplaced; the dendrons of the ganglion cell in the center are quite numerous, swollen and densely stained. Toluidin blue; × 250.

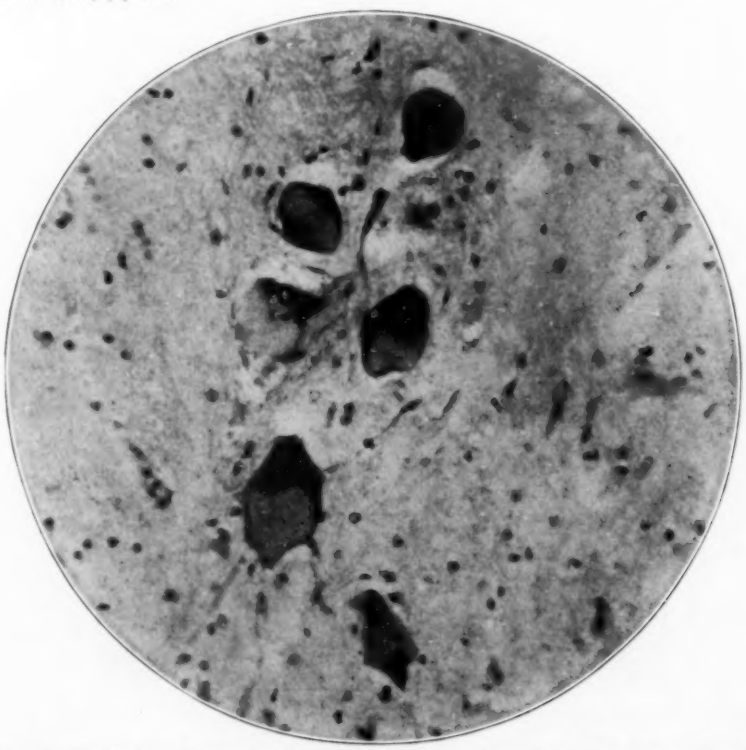

Fig. 4.—Spinal cord—ventral horns: the ganglion cells show an endocellular network in the basal portion which is devoid of Nissl's substance (hand lens should be used). Toluidin blue; × 230.

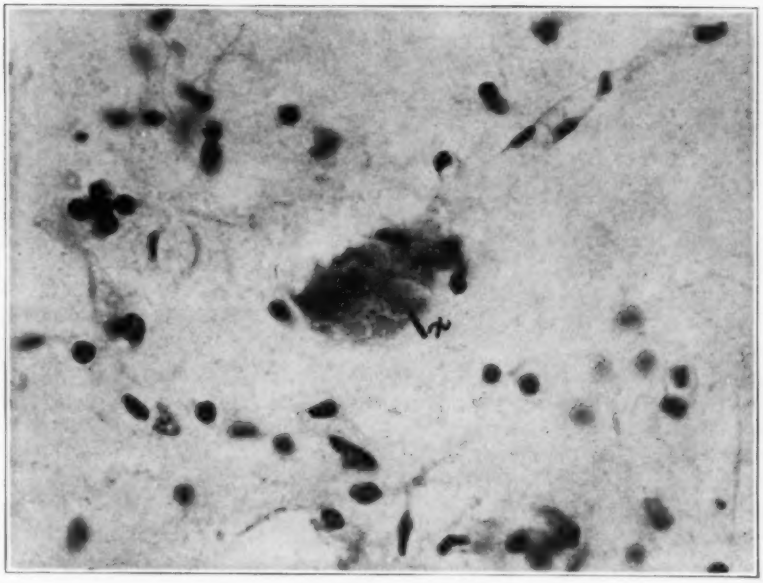

Fig. 5.—Substantia nigra: the center of the photomicrograph shows a swollen and liquefied ganglion cell; at *x* is a vacuole containing an amorphous substance (hand lens should be used). Toluidin blue; × 560.

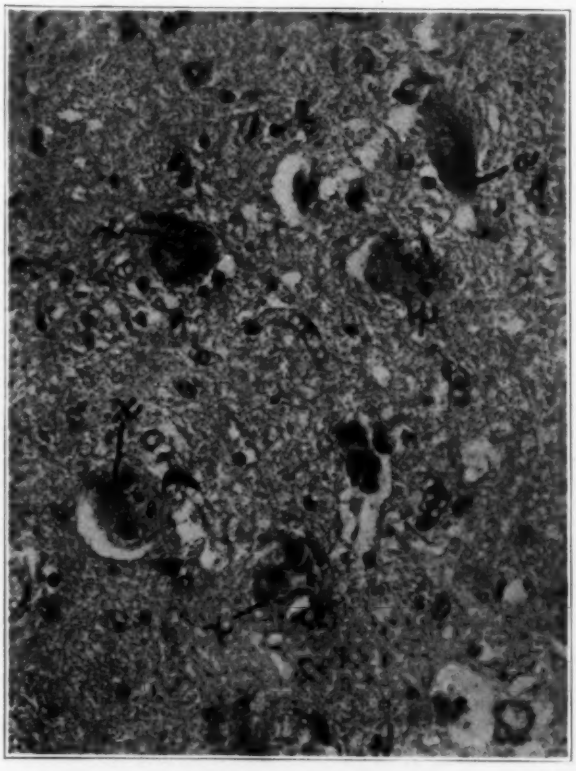

Fig. 6.—Nucleus ruber: numerous enclosures (*x* and *a*) within the ganglion cells, described in text. Bielschowsky stain counterstained with Alzheimer-Mann; × 350.

in size, pyknotic, misplaced, occasionally disfigured and shrunken. Such cells were especially common in the occipital lobe, Ammon's horn and the pons.

A third group of ganglion cells showed still more noteworthy changes. As figure 5 shows, the cell body is distended, swollen and filled with a dustlike substance and contains a vacuole enclosing an amorphous substance (at x). Other cells contained several oval or round vacuoles (fig. 6) within their cytoplasm filled with small particles of an amorphous substance, which showed no capsule or concentric markings and stained brilliant green with toluidin blue, yellowish with van Gieson's stain and hematoxylin-eosin, bluish with Alzheimer-Mann's mixture, brownish yellow with the Bielschowsky stain, pinkish red with nile blue and, what is rather noteworthy, red with scarlet red. They did not give the reactions for amyloid, nor did they dissolve in ether, alcohol or xylol. Such cells were especially numerous in the substantia nigra and nucleus ruber, but were also scattered throughout the cortex, optic thalamus and other portions of the central nervous system. Often such cells retained their configuration fairly well, possessed a fair amount of Nissl bodies and were supplied with a large number of dendrons, densely stained and somewhat swollen. Other cells, as already pointed out, were enormously distended, elongated and reticular, and often exhibited phenomena of satellitosis and neuronophagia.

Finally, some cells showed marked chromatolysis and shrinkage; the nucleus was dark and the processes were well stained and tortuous; some appeared liquefied or reduced to mere shadows. Such cells were quite common in the nucleus ruber.

The foregoing varieties of cell changes—expanded cell bodies with no or few dendrons, shrunken cells with pyknotic nuclei, vacuolated cells filled with granules, and apparently normal ganglion cells with a fair amount of Nissl bodies—were encountered also in the optic thalamus. However, the far-gone cell destruction, such as is seen in the infantile type of amaurotic family idiocy, with the formation of mulberry bodies and gitter cells was not seen in the optic thalami of this case.

The changes outlined were also present in the cerebellum, which, as already stated, was greatly reduced in size. As figure 7 shows, all the layers—molecular, that of Purkinje cells and granular—were atrophied. There was great poverty not only in cells but also in nerve fibers. Thus the Purkinje cells were, in the majority of lamellae, diminished in number; some lamellae showed very few, some again showed more, but no lamellae containing a normal number of Purkinje cells were found. Without exception these appeared naked and destitute of the basket fibers (fig. 8), which are so abundant in a normal cerebellum (fig. 9). In Bielschowsky specimens the endocellular network was well developed, the long fibrils occupying the surface of the cell body and extending into the apical dendron.

Aside from purely morphologic changes, the ganglion cells exhibited also microchemical changes. Thus, scarlet red and sudan III staining methods exhibited small granules of orange yellow color which packed the basal portion of the cell body, causing its bulging with displacement of the nucleus to the periphery. On superficial examination and under a low power such stained cells sometimes appeared (in the occipital lobe, Ammon's horn and cerebellum) homogeneous. In other regions, such as the optic thalamus, the granules appeared somewhat larger and bright red, crowding and overlapping each other and enclosed within the vacuoles of the endocellular network. Hematoxylin staining methods of Weigert-Kulschitzky and Schaffer did not reveal in them the type of granules which are so abundant in the infantile form of amaurotic family idiocy, while the methods of Heidenhain-Spielmeyer showed granules—minute and scanty—only in some instances. Instead, the cells so stained commonly appeared diffusely gray while

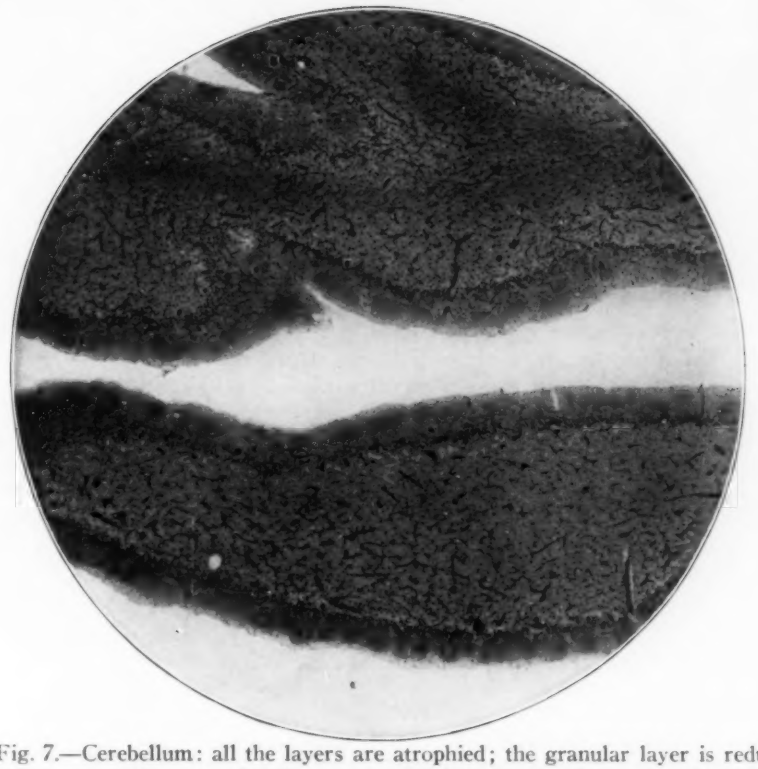

Fig. 7.—Cerebellum: all the layers are atrophied; the granular layer is reduced in size to a single row of cells among which, with the help of a hand lens, Purkinje cells can be seen, also reduced in number and size; the medullary portion (the white substance) is enormously vascularized. Ramón y Cajal stain; × 33.

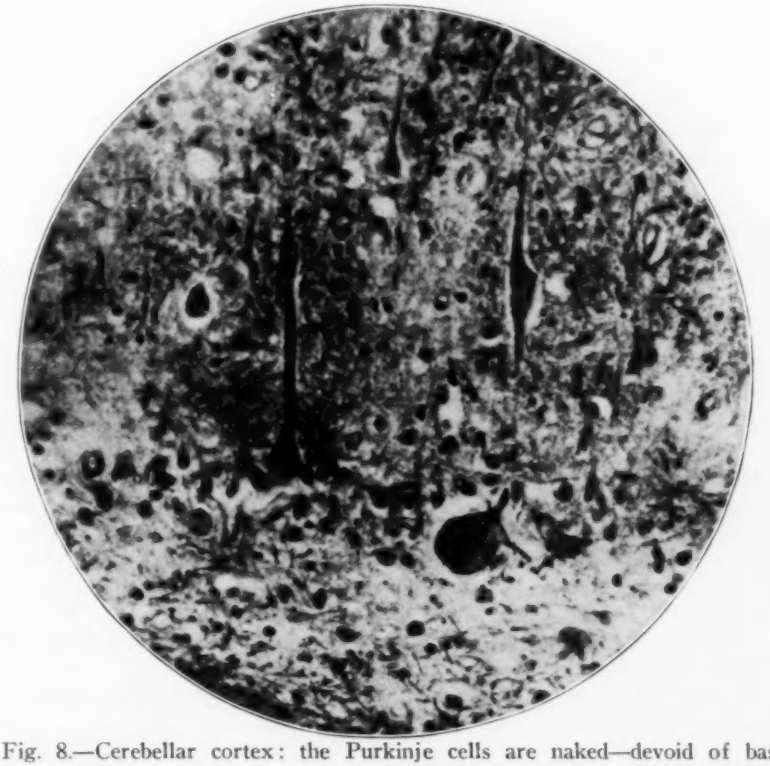

Fig. 8.—Cerebellar cortex: the Purkinje cells are naked—devoid of basket fibers; the one to the right is markedly swollen and exhibits an endocellular network; scattered over the visual field are a number of swollen dendrons; the granular layer is poor in ganglion cells and devoid of nerve fibers (comparison should be made with figure 9). Bielschowsky stain; × 325.

the spinal cord cells appeared entirely colorless. Neither did osmic acid show black granules anywhere in the brain or spinal cord. Only with Cajal's and del Rio Hortega's, and occasionally with Bielschowsky's, silver staining methods were the ganglion cells of some areas (occipital lobe, Purkinje cells) incrusted with numerous fairly large dark granules. In other areas only a scanty amount of granules were present within a homogeneous cell substance. In short, some areas showed an abundance of argentophil granules, while hematoxynophil and what Schaffer termed osmioreductive granules were absent. The cell contents in this case may generally be classified as lipoids and prelipoids of Alzheimer. The lipoids are also referred to (Dide, Guiraud and Michel,[6] Hurst[7]) as cerebrosides

Fig. 9.—Normal cerebellum: basket fibers around the Purkinje cells and the wealth of ganglion cells in the granular layer are well seen (comparison should be made with figure 8). Stain and magnification the same as in figure 8.

(fatty acid combinations with radicles conaining nitrogen and carbohydrate) and phosphatides (combinations of fatty acids with radicles containing nitrogen and phosphorus). Bielschowsky considers the ganglion cell lipoid contents in the late infantile type to be lecithinoid substances, while Marinesco holds that they represent rather a mixture of various fatlike substances which originate

6. Dide, M.; Guiraud, P., and Michel, R.: Lésions nerveuses dans un cas de maladie de Tay-Sachs juvenile, Encéphale **15**:303 (May) 1920.

7. Hurst, E. W.: A Study of the Lipoids in Neuronic Degeneration in Amaurotic Family Idiocy, Brain **48**:1 (March) 1925.

Fig. 10.—Medulla oblongata: the pyramids, olivocerebellar fibers, the ninth and twelfth nerves, the corpora restiformia, etc., show no changes. Weigert-Pal; × 5.

Fig. 11. Normal peduncles, except the frontopontile tract (*F. p.*) which is atrophied; the adjacent bundles of the third nerve are well preserved; the white substance of the red nucleus is atrophied. Weigert-Pal; × 4.

from mitochondria, fuchsinophile granules of neurosomes. Whatever the nature and origin of the granular contents as well as of the enclosures pictured in figures 5 and 6, they denote a profound disturbance in the cell metabolism and may generally be looked on as the "Abbauprodukte" of Alzheimer.

Notwithstanding the manifest structural and chemical changes of the ganglion cells, satellitosis and neuronophagic phenomena were rather rare; nor did the axons appear tumefied or show signs of degeneration. The white substance, therefore, was practically normal though rarefied. Thus the various columns of the spinal cord, the anterior and posterior roots, Clarke's columns, the spinocerebellar tracts, the medulla oblongata (fig. 10), including the olivary bodies and the accessory olives, the restiform bodies, the pons, the superior and middle cerebellar peduncles, the dentate nucleus—all appeared normal. Only the frontopontile fibers (*F. P.*, fig. 11) and the medullary fibers of some cerebellar folia (fig. 12), especially in their crests, were markedly rarefied while the tangential fibers were completely lacking. These were also absent throughout the cortex of the brain. Rarefaction of myelin fibers was also in evidence in the optic tracts where some of the preserved axons appeared rather thick, with mild reactive glial and mesodermal tissue phenomena. Nor did the subcortical white medullary substance, studied with various methods, or the nerve fibers of the bulbar nerves (figs. 10 and 11) exhibit signs of secondary degeneration, a rather remarkable fact in the presence of such widespread structural and chemical cell change. Glial reactive phenomena, for instance, were rather inconspicuous. The glia, as a rule, appeared as large oval or round nuclei, rich in chromatin, devoid of cytoplasm and occasionally forming clusters. Progressive glia phenomena with formation of fibers were present only in some regions—occipital lobe, cerebellum and optic thalamus. As figure 2 shows, a marked glial activity has been going on in the third, fourth and fifth layers of the occipital lobe. Here were seen (fig. 13) large chromatin-rich glia nuclei within an abundant cytoplasm emitting thick, powerful ramifying processes which envelop the blood vessels. Gitter cells packed with lipoids were quite numerous, scattered over the visual field which was made up of a dense feltwork of thin glia fibers. Marked proliferative glia phenomena were also in evidence in the stratum zonale and the subependymal glia of the lateral ventricles.

Mesodermal Elements.—The blood vessels for the most part exhibited no inflammatory or degenerative phenomena. Exceptionally, a capillary was seen infiltrated with lymphocytes and gitter cells, the latter being the most common enclosures of the adventitial spaces throughout the brain and spinal cord.

In contrast to the ganglion cell contents which, as said, stained orange yellow with scarlet red, those of the adventitial spaces stained bright red. Often, new formed capillaries were seen with hypertrophied adventitial cells, while with the staining methods of Holzer, Alzheimer-Mann and especially those of Ramón y Cajal and del Rio Hortega, enormous numbers of blood vessels covered the visual field, principally in the cerebellum (fig. 7), caudate nucleus and occipital lobe.

Choroid Plexus.—The tuft cells of the lateral ventricles were pale, round, somewhat expanded, and granular, much resembling gitter cells; their nuclei were well stained and centrally located. The stroma was proliferated, containing numerous fibroblasts, corpora arynacea and greatly distended and hyperemic blood vessels.

Pia.—This showed distended meshes enclosing fibroblasts and oblong or oval cells containing one or two nuclei, sometimes horseshoe shaped and quite pale (polyblasts). Other cell bodies were represented by markedly pyknotic nuclei, or appeared granular with an eccentric nucleus and packed with fatty substances (gitter cells), while other cells, quite numerous, were mesothelial cells that

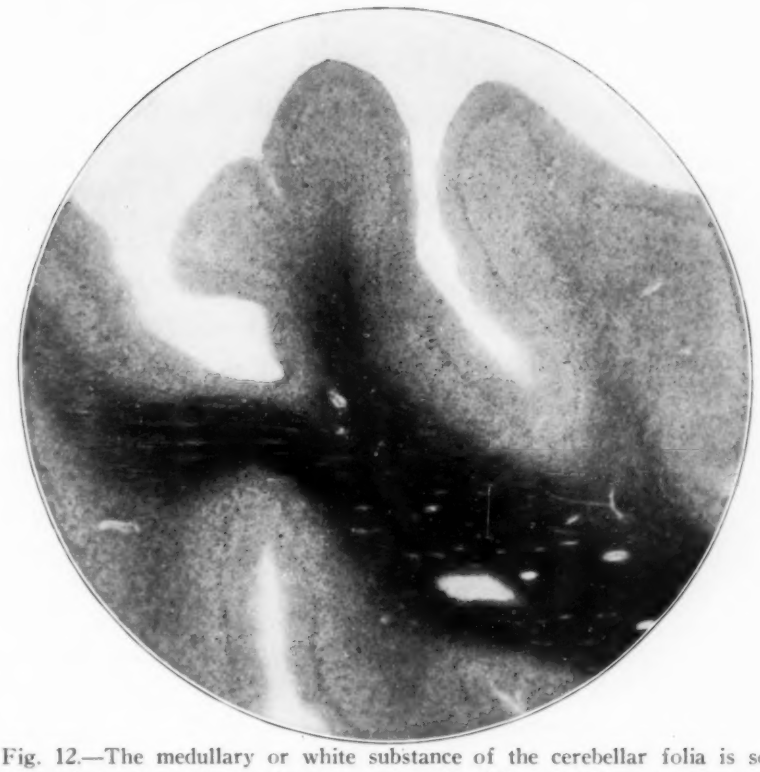

Fig. 12.—The medullary or white substance of the cerebellar folia is somewhat rarefied and practically absent in one to the right. Spielmeyer's hematoxylin stain; × 24.

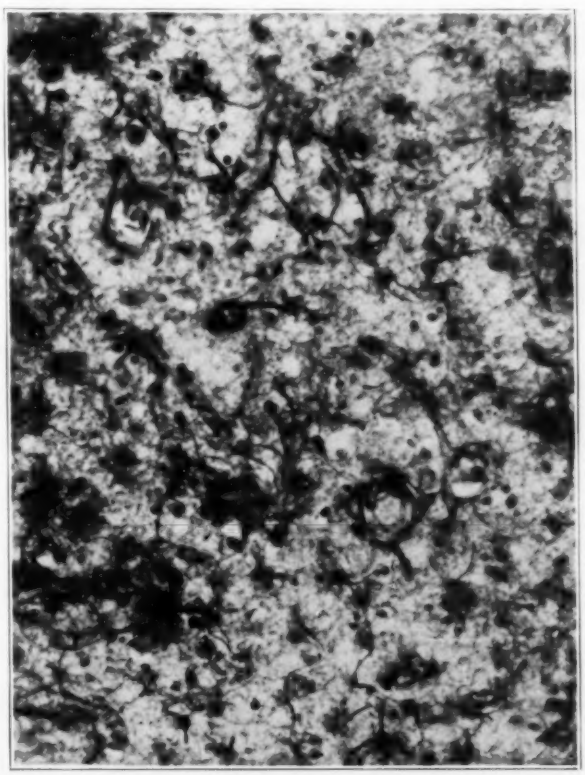

Fig. 13.—Fibrous glia in the lower strata of the occipital lobe. Higher magnification of figure 2. Holzer's glia stain; × 230.

usually formed clusters. In addition, there were macrophages containing granular pigment, mast cells and numerous corpora arynacea (around the cerebellum). The blood vessels of the pia were hyperemic but exhibited no inflammatory phenomena.

SUMMARY OF THE PATHOLOGIC CHANGES

The pathologic changes included widespread cellular changes typical of amaurotic family idiocy; vacuolated cells containing amorphous deposits; atrophy of all cerebellar layers; rarefaction of medullary nerve fibers; marked degeneration of the deeper layers of the occipital lobe, and of the ganglion cells of the nucleus ruber; absence of secondary degeneration of the white substance of the brain and spinal cord; lipoid accumulation in the adventitial spaces of the blood vessels and in the subarachnoid space; great scarcity of inflammatory phenomena.

COMMENT

In the type of structural changes of the ganglion cells, the case under discussion resembled the classic infantile type of amaurotic family idiocy; in the late onset (at age of 3½ years), protracted course (four years), the presence of cerebellar atrophy, the absence of the racial element and of the macular changes, it resembled the type described by Bielschowsky as late infantile, or by Jansky as cerebellar; while in some cellular changes it rather resembled the juvenile type of Vogt-Spielmeyer. However, in the latter the cell deformities are much less pronounced than in the classic Tay-Sachs' form and the course is even more protracted than in the late infantile, cerebellar type of Jansky-Bielschowsky. The remarkable feature, however, is not so much the morphologic appearance of the changed ganglion cells as their microchemical changes. In the infantile type, the ganglion cells, as a rule, do not stain bright red with scarlet red, but do stain readily with the hematoxylin methods of Weigert and of Schaffer. Only in the optic thalamus some cells give the specific stains with scarlet red, sudan III and even osmic acid, indicating, as pointed out elsewhere,[8] that the optic thalami are most intensively and extensively involved in the infantile type of amaurotic family idiocy. That is to say, nowhere does the central nervous system show in the latter such an advanced massive cell destruction as in the optic thalami, though isolated, scattered, ganglionic changes occur also in other regions. In the juvenile type of Vogt-Spielmeyer the contents of the ganglion cells, in contrast to what is seen in the infantile form, do not stain with Weigert's or with Schaffer's method, but usually do stain well with Heidenhain-Spielmeyer's method, osmic acid and scarlet

8. Hassin, G. B.: A Study of the Histopathology of Amaurotic Family Idiocy (Infantile Type of Tay-Sachs), Arch. Neurol. & Psychiat. **12**:640 (Dec.) 1924.

red. In the case here recorded neither of the latter staining methods affected the cell contents, which stained but slightly yellow-orange with scarlet red or sudan III. Such a difference in the microchemical reactions may be due to the stage of the disease process which, being more advanced in the late infantile and juvenile forms, reflects on the chemical properties of the ganglion cells in general. As a rule the chemical changes, as discussed elsewhere,[8] correspond with the type of the idiocy, though there are exceptions. For instance, in the cases of both Westphal,[9] infantile type, and Schob,[2] late infantile type, the cells stained bright red with the Herxheimer stain, while in the juvenile cases of Kufs [10] and Walter,[11] they stained with the Herxheimer stain only light red, but intensely black with osmic acid (especially the case of Walter). Cases like these are of an unusual interest and value, for they embody features that are peculiar for the three types of what generally might be considered one disease process.

The cell contents in the present case offered another peculiarity—enclosures within vacuoles. As pointed out, these were irregular in shape and often resembled amyloid bodies without, however, giving the specific reactions. Cell enclosures, aside from those known as lipoids and prelipoids, are generally of rare occurrence. They have been described by Bevan Lewis [12] in epileptic idiocy as "colorless or highly translucent oval or spheroidal bodies;" by Lafora [13] and Westphal [14] in myoclonus epilepsy as amyloid bodies; by Bielschowsky [5] in a case of late amaurotic family idiocy as homogeneous globular enclosures with a somewhat lightly stained center (however, he does not consider them pathologic); by Schob [2] in a case of amaurotic family idiocy (a boy, aged 8) as "round or polygonal bodies, somewhat blue or yellowish green, depending on differentiation," and by Spielmeyer [15] who, in a case of degenerative atrophy of the upper extremities associated with marked speech defects, found the cortical and anterior horn cells swollen and contain-

9. Westphal, A.: Beitrag zur Lehre von der amaurotischen Idiotie, Arch. f. Psychiat. **58**:248, 1917.

10. Kufs, H.: Ueber eine Spätform der amaurotischen Idiotie und ihre heredofamiliären Grundlagen, Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:169, 1925.

11. Walter, F. K.: Ueber familiäre Idiotie, Ztschr. f. d. ges. Neurol. u. Psychiat. **40**:349, 1918.

12. Lewis, Bevan: Notes on Certain Lesions of the Nervous Tissues of Frequent Occurrence in the Brains of the Insane, Brain **2**:364, 1879-1880.

13. Lafora, G. R.: Ueber das Vorkommen amyloider Körperchen im Innern der Ganglienzellen, Virchows Arch. f. path. Anat. **205**:295, 1911.

14. Westphal, A.: Ueber eigenartige Einschlüsse in den Ganglienzellen (corpora amyblacea) bei einem Falle von Myoclonus-Epilepsie, Arch. f. Psychiat. **60**: 769, 1919.

15. Spielmeyer, W.: Zur Frage der sogenannten spezifischen Ganglienzellerkrankungen, Ztschr. f. d. ges. Neurol. u. Psychiat. (Referate) **5**:967, 1912.

ing a substance that stained with methyl violet, but with no other reagents for amyloid. The nature of the enclosures described has not been established. Some, as Lafora and Westphal, considered them amyloid, some (Bevan Lewis) colloidal substances. The latter was most probable in my specimens, though morphologically they, in some instances, much resembled amyloid bodies and chemically lipoids. Yet they were not affected by such chemicals as alcohol, xylol, ether.

Of equal interest were some focal anatomic changes in the central nervous system. Of these the most outstanding was the cerebellar atrophy. This has been noted in every case of the late infantile form of amaurotic family idiocy recorded, and Jansky,[16] therefore, suggested for this type the name "cerebellar." The varieties of cerebellar atrophy are classified by Bielschowsky [5] as cerebellofugal (efferent) and cerebellopetal (afferent). The former, represented in his opinion by Marie's hereditary cerebellar ataxia, is characterized by the lack of Purkinje cells and their axons. The basket fibers enclosing the cells are therefore empty and, like the mossy and climbing fibers, well preserved. In the centripetal type, the granular layer, the basket, climbing and mossy fibers, that is to say, the whole centripetal system is lacking, but the Purkinje cells are partly preserved. The centripetal type of cerebellar atrophy occurs, according to Bielschowsky, in the late infantile type of amaurotic family idiocy in which he also found atrophy of the dentate and other cerebellar nuclei with degeneration of the olivocerebellar tract, the middle cerebellar peduncles being pale and reduced in size. The anatomic changes in my case somewhat correspond with those of Bielschowsky, artificial as his classification might appear. Thus all the mossy, basket, climbing and tangential fibers were affected, but the olivocerebellar tract (fig. 10) and the middle and other cerebellar peduncles were intact.

Remarkable as the constant occurrence of the cerebellar atrophy is, it cannot be considered pathognomonic for the late infantile form, as it has also been found in the infantile form (cases of Westphal,[9] of Dollinger,[17] Globus,[18] Ostertag [19]) and the juvenile forms (Kufs' and Globus' cases). Though cerebellar atrophy in the last two conditions

16. Jansky, J., and Mysliveček, F.: Beitrag zur familiären amaurotischen Idiotie, Arch. f. Psychiat. **59**:668, 1919.

17. Dollinger, A.: Zur Klinik der infantilen Form der familiären amaurotischen Idiotie (Tay-Sachs), Ztschr. f. Kinderh. **22**:167, 1919.

18. Globus, J.: Ein Beitrag zur Histopathologie der amaurotischen Idiotie, Ztschr. f. d. ges. Neurol. u. Psychiat. **85**:424, 1923.

19. Ostertag, B.: Entwickelungstörungen des Gehirns und zur Histologie und Pathogenese, besonders der degenerativen Markerkrankung bei amaurotischer Idiotie, Arch. f. Psychiat. **75**:355, 1925.

is rare and mild, it denotes, like the microchemical properties of the ganglion cells, their common origin, a membership in one disease process.

The ganglion cell changes outlined—structural and microchemical—were responsible for the clinical picture in this case. The outstanding clinical feature was decerebrate rigidity as experimentally produced in animals by Sherrington.[20] However, there is some difference between the experimental and the human decerebrate rigidity. In the former, the elbows like the legs were extended or straightened, the wrists were somewhat flexed and the hand (of the monkey) was turned with the palmar surface somewhat inward; in the lower extremity the extension also affected "somewhat" the ankle. Ablation of the cerebellum did not abolish the rigidity, but the limbs at once became flaccid if, after ablation of both cerebral hemispheres, the afferent roots previously laid bare and prepared were carefully severed. The result was quite local, that is, confined to the one limb, the afferent roots of which were severed.

These phenomena discovered by Sherrington in animals are but partially observed in human decerebrate rigidity in which we deal with flexion-extension rigidity; that is, the upper extremities are semiflexed and the feet are in plantar flexion. The other difference is the time element—experimental decerebrate rigidity is essentially acute, while the human form is essentially chronic, though Bazett and Penfield [21] described Sherrington phenomena in experimental animals with a chronic course. With the discovery by Magnus and de Kleijn of the neck and labyrinthine reflexes, laboratory as well as clinical reports on decerebrate rigidity multiplied immensely. However, while experimentally produced decerebrate rigidity was quite uniform and fairly typical, that presented by some clinical cases was not typical at all. In many cases it was incomplete and fragmentary. Walshe [22] justly pointed out that some, if not the majority, of clinical cases recorded as decerebrate rigidity, actually do not belong here. He considers essential for this condition the quality of rigidity—a true hypertonus, with prolongation of the tendon reflexes, clasp-knife spasticity—in short, the presence of pyramidal tract involvement is, according to Walshe, an essential factor in the production of decerebrate rigidity in animals and of spasticity in man. He eliminates as a factor the extrapyramidal motor system which he assumes has no relation whatever with decerebrate rigidity. The

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20. Sherrington, C. S.: Decerebrate Rigidity and Reflex Coordination of Movements, J. Physiol. **22**:319, 1897-1898; The Integrative Action of the Nervous System, New York, Charles Scribner's Sons, 1906, p. 299.

21. Bazett, H. C., and Penfield, W. G.: A Study of the Sherrington Decerebrate Rigidity in the Chronic as Well as the Acute Condition, Brain **45**:185 (Oct.) 1922.

22. Walshe, F. M. R.: The Decerebrate Rigidity of Sherrington in Man, Arch. Neurol. & Psychiat. **10**:1 (July) 1923.

clinical studies of Simons [23] seem to support the views of Walshe, while those of McAlpine,[24] of Brock and Wechsler [25] do not. The few cases in which decerebrate rigidity has been studied, both clinically and pathologically, showed a variety of pathologic conditions with and without pyramidal tract involvement. Thus Simons observed the presence of Magnus-de Kleijn neck, but not labyrinthine, reflexes in many cases of organic hemiplegia due to a pyramidal tract lesion. In cases with extrapyramidal, but without pyramidal, signs and symptoms, such as hypertonia, spastic reflexes, hemiplegic associated movements, the neck reflexes of Magnus-de Kleijn could never be elicited. On the other hand, a great variety of pathologic conditions were recorded in which decerebrate rigidity could not be ascribed to the lesion of the pyramidal system, that is to say, it occurred in its absence. Here belong the cases of Magnus and de Kleijn,[26] de Bruin,[27] Dollinger,[17] Marinesco and Rudovici,[28] and Ostertag [19]—all of amaurotic family idiocy; of Brouwer [29]—a case of diffuse meningo-encephalitis with destruction of the white substance of the brain and the cerebellum; of Leon Meyers [30]—two cases of degeneration of the central nervous system and its maldevelopment; of Walshe [31] and Loyal Davis [32]—suprapituitary tumors; of Weiland [33]—a case of an obscure, possibly syphilitic, lesion of the brain (not verified by a postmortem examination), and of Magnus [34] in a child, aged 9 days, with a hemorrhage in both lenticular nuclei. Of the

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23. Simons, A.: Kopfhaltung und Muskeltonus, Ztschr. f. d. ges. Neurol. u. Psychiat. **80**:499, 1923.

24. McAlpine, D.: A Clinical Study of Plastic Tonus as Observed in a Rare Sequela of Epidemic Encephalitis, Brain **47**:178 (May) 1924.

25. Brock, S., and Wechsler, I. S.: Involuntary Movements; Their Unusual Association and Relation to the Phenomena of Decerebrate Rigidity, Arch. Neurol. & Psychiat. **11**:698 (June) 1924.

26. Magnus, R., and de Kleijn, A.: Weitere Beobachtungen über Hals- und Labyrinthreflexe auf die Gliedermuskeln des Menschen., Pflüger's Arch. f. d. ges. Physiol. **160**:429, 1914 (case 8).

27. De Bruin, J.: Abstr., Jahrb. f. Kinderh. **81**:461, 1915.

28. Marinesco, G., and Radovici, A.: Idiotie amaurotique et rigidité décérébrée, Encéphale **18**:145 (March) 1923.

29. Brouwer, B.: Ueber Meningo-Encephalitis und die Magnus-de Kleijnschen Reflexe, Ztschr. f. d. ges. Neurol. u. Psychiat. **36**:161, 1917.

30. Meyers, L.: Magnus and De Kleijn Phenomenon in Brain Lesions, Arch. Neurol. & Psychiat. **8**:383 (Oct.) 1922.

31. Walshe, F. M. R.: Complete Decerebrate Rigidity in Man, Lancet **2**:644 (Sept. 29) 1923.

32. Davis, Loyal: Decerebrate Rigidity in Man, Arch. Neurol & Psychiat. **13**:569 (May) 1925.

33. Weiland: Die Abhängigeit der Stellung der Glieder vom Kopf, München. med. Wchnschr. **59**:2539 (Nov. 12) 1912.

34. Magnus, R.: Ueber die Beziehungen des Kopfes zu Gliedern, München. med. Wchnschr. **59**:681 (March 26) 1912.

foregoing pathologic conditions amaurotic family idiocy is thus the most common in which decerebrate rigidity occurs. If one considers the fact that some phases of decerebrate rigidity, such as extensor spasticity for instance, has been mentioned in a great many cases of amaurotic family idiocy, one must come to the conclusion that this morbid condition is the most potent and common etiologic factor in decerebrate rigidity in man. Like the cherry spot in the classic type of Tay-Sachs, the presence of decerebrate rigidity may serve as a means for the diagnosis of other types of this disease in which the cherry spot is absent and which, therefore, remain unrecognized.

Pathologic studies of such cases may even give some idea as to the rôle some portions of the brain play in its causation. For instance, the atrophy of the cerebellum, which may be considered equivalent to its experimental ablation, had no effect in my case on the decerebrate rigidity. This is in accord with Sherrington's discovery that the latter does not disappear in decerebellate animals. It also confirms the observation of Magnus [35] that ablation of the cerebellum (in cats) does not inhibit the neck or labyrinthine reflexes. On the other hand it seems to be at variance with what occurred in the experiments of Weed,[36] who in the majority of his acute and subacute cases found the rigidity to disappear in decerebellate animals. Weed and others also found that preservation of the nucleus ruber is essential in the successful production of decerebrate rigidity. In my case the nucleus ruber has been probably more damaged (figs. 6 and 11) than the cerebellum itself, yet it did not affect the rigidity.

The remarkable preservation of the white fibers, including the pyramidal system, seems to disprove the contention of Walshe that the latter is the essential factor in the production of decerebrate rigidity, though the possibility of some functional incapacity of the pyramidal fibers cannot be excluded because of the changed structure of the cortical cell elements.

Of additional pathologic interest is the marked atrophy of the cerebellum. This was symmetrical, involving all the layers; it was primary, that is to say, it was not caused by a hemorrhage, a softening or a lesion of the spinocerebellar tract. It was purely degenerative, with secondary proliferation of the neuroglia and marked vascularization. We probably are dealing here first with aplasia of the cerebellum, or rather of the cerebellar ganglion cells which ultimately become degenerated. As shown in my previous contribution,[8] in the classic amaurotic family idiocy we

35. Magnus, R.: Welche Theile des Zentralnervensystems müssen für das Zustandekommen der tonischen Hals- und Labyrinthreflexe auf die Körpermuskulatur vorhanden sein? Pflüger's Arch. f. d. ges. Physiol. **159**:224, 1914.

36. Weed, L. H.: Observations upon Decerebrate Rigidity, J. Physiol. **48**:205, 1914.

also deal with maldevelopment of the ganglion cells and in some cases with that of the white substance followed by parenchymatous degenerations, especially of the optic thalami. These, in the late infantile form, exhibit changes which are comparatively mild. This is in contrast to what is found in the cerebellum in which the white substance is fairly well preserved in the presence of a general maldevelopment of the ganglion cells. One might therefore assume that the pathologic changes, in the infantile form of amaurotic family idiocy, are mainly, though not exclusively, in the optic thalamus; in the late infantile type they are mainly in the cerebellum.

As to the clinical features, I wish to point out that the patient's attitude in exhibiting the Magnus-de Kleijn neck reflex strongly resembled the one occurring during the tonic phase of an epileptic seizure: the same extension rigidity of the extremities toward which the head is turned, with plantar flexion of the feet and toes, adduction and semiflexion of the elbows, hands and fingers of the opposite side, and retraction of the head. We might say that in an epileptic attack we deal with decerebrate rigidity or that the latter is but a phase of a modified epileptic seizure. It is quite probable that the mechanism underlying the neck reflex of Magnus-de Kleijn in a decerebrated animal and as it occurs during an epileptic attack is the same. In the former the abnormal position of the neck resulting in alternating changes in the flexors-extensors of the extremities is caused by the experimenter; in epilepsy by some invisible force. The mere fact that in an epileptic seizure a condition of decerebrate rigidity obtains, with temporary suspension of the cortical functions, denotes that the cerebral cortex or the larger portion of the brain is not instrumental in the production of an epileptic attack; that the "seat" of epilepsy is not in the cortex. It is rather significant that both epilepsy and decerebrate rigidity are exceedingly common in amaurotic family idiocy and that both may show here as incomplete or partial phenomena: the former as clonic fits (so marked in my patient and in the patients of Magnus-de Kleijn and Ostertag), the latter as extensor spasticity.

One more extraordinary feature of this remarkable case was the occurrence of the disease in one of twins. Only two other such incidents are mentioned in the literature, one by Herrman [37] and one by Sachs.[38] Both cases were of the infantile variety.

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37. Herrman, Charles: A Case of Amaurotic Family Idiocy in One of Twins, Arch. Pediat. **32**:902, 1915.

38. Sachs, B.: The Heredity Factors Operative in Amaurotic Family Idiocy, in Heredity in Nervous and Mental Diseases, Association for Research in Nervous and Mental Disease, New York, Paul B. Hoeber, **3**:159. Sachs, B., and Hausman, L.: Nervous and Mental Disorders from Birth Through Adolescence, New York, Paul B. Hoeber, 1926, p. 308.

CONCLUSIONS

1. In addition to the well-known classic type of amaurotic family idiocy, known as infantile or Tay-Sachs disease, a late infantile and juvenile forms are established.

2. The three varieties differ in the age of onset, the course and the absence (in the last two types) of macular changes and of the racial element.

3. Pathologically resembling the infantile form, the late infantile type differs in the existence of marked cerebellar changes which are unusual in the former.

4. The optic thalamus changes, so marked in the infantile form, are here much less in evidence.

5. Decerebrate rigidity occurs in amaurotic family idiocy more frequently than in any other organic brain lesion and should therefore suggest this morbid condition in obscure cerebral lesions (at least in children).

6. The phenomena of the Magnus-de Kleijn neck reflex much resemble those occurring in the tonic phase of an epileptic seizure.

7. Amaurotic family idiocy may affect only one of twins, sparing the other.

ANEURYSMS OF CEREBRAL VESSELS

CLINICAL MANIFESTATIONS AND PATHOLOGY *

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The correct diagnosis of a cerebral aneurysm during the life of the patient has been regarded as somewhat difficult. Earlier writers on the subject were even doubtful as to whether a diagnosis could be made. In 1859, Gull [1] gave expression to this pessimism by writing: "Although we may from the circumstances sometimes suspect the presence of an intracranial aneurysm, we have at the best no symptom upon which to ground more than a probable diagnosis." In 1916, more than half a century later, with all the additional knowledge of the intervening years, Fearnsides [2] considered that this dictum still held true. However, this does not mean that we have ceased to gather knowledge in regard to the clinical features of the disease, especially with the advancement of neurosurgery and the consequent increasing interest in intracranial pathology. Our therapeutic horizon has grown wider, and this has given a tremendous impetus to the study of intracranial disease. Surgical exploration of the cranial cavity has more than once revealed a pulsating saccular aneurysm where a neoplasm was expected. The study of the subject is something of more than academic importance in the differential diagnosis of intracranial disease.

INCIDENCE

Mention of saccular cerebral aneurysm is not common in clinical literature. It is in the necropsy records that cases are more easily found. The literature dealing with records of cases diagnosed as aneurysm during life is rather scanty. Symonds [3] recently published records of five cases in which he had made a diagnosis of cerebral aneurysm during life and was able later to confirm his diagnosis at necropsy in three instances. Cushing [4] was able to collect records of

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* Read before the Section on Neurology at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

1. Gull, W. W.: Cases of Aneurysm of the Cerebral Vessels, Guy's Hosp. Rep. **5**:281-304, 1859; A Collection of the Published Writings of William Withey Gull, London, The New Sydenham Society, 1894, pp. 86-106.

2. Fearnsides, E. G.: Intracranial Aneurysms, Brain **39**:224-296 (Oct.) 1916.

3. Symonds, C. P.: Contributions to the Clinical Study of Intracranial Aneurysms, Guy's Hosp. Rep. **73**:139 (April) 1923.

4. Cushing, Harvey: Contributions to the Clinical Study of Intracranial Aneurysms, Guy's Hosp. Rep. **73**:159 (April) 1923.

only four cases of cerebral aneurysm in his own experience; in two of these operation had been performed, in one by himself and in the other by Elsberg. All four aneurysms had originated near the bifurcation of the internal carotid. In the two cases the symptoms were surprisingly like those due to neoplasm, and actually the aneurysms had not ruptured but had given the signs associated with an expanding body in the cranial cavity. For the rest, the literature deals mainly with studies of collections of individual reports of verified cases and series of cases observed in the necropsy room and traced in retrospect. Fearnsides' series is an example of the latter method of study. His series was obtained from the records of the Pathological Institute of the London Hospital, between 1907 and 1913. During this period, there had been 7,924 necropsies and in 5,432 of these an examination of the head had been permitted. Saccular cerebral aneurysms were found in forty-four instances. In four of these, multiple aneurysms were present. Osler,[5] in his first 800 necropsies in Montreal, found them in twelve instances. Beadles [6] gathered 555 records of death from cerebral aneurysm, 114 of which were records of new cases. The pathologist has much more experience with this disease than the clinician. Records of clinical experience of aneurysm are so few chiefly because cerebral aneurysm is seldom thought of and only occasionally recognized during the lifetime of the patient.

ETIOLOGIC AND PATHOLOGIC CHARACTERS

The only type of aneurysm under discussion at present is the focal true fusiform or saccular aneurysm easily visible to the naked eye. Miliary aneurysms are in a somewhat different category and, while they were originally regarded by Charcot [7] as the sine qua non of cerebral hemorrhage, the more recent work of Pick,[8] Shennan,[9] Raeder [10] and others has shown that their frequency and importance have been greatly overrated. Infiltration of blood and fibrin into the walls of the vessel,

5. Osler, William: The Principles and Practice of Medicine, ed. 8, New York, D. Appleton & Co., 1912, p. 982.

6. Beadles, C. F.: Aneurysms of the Larger Cerebral Arteries, Brain **30**: 285-336, 1907.

7. Charcot, J. M., and Bouchard, C.: Nouvelles récherches sur la pathogénie de l'hémorrhagie cérébrale, Arch. de physiol. norm. et path. **1**:110-127, 1868.

8. Pick, L.: Ueber die sogenannten miliaren Aneurysmen der Hirngefässe, Berl. klin. Wchnschr. **47**:325-329, 1910.

9. Shennan, Theodore: Miliary Aneurysms, in Relation to Cerebral Hemorrhage, Edinburgh M. J. **15**:245-252, 1915.

10. Raeder, O. J.: Repeated Multiple Minute Corticospinal Hemorrhages with Miliary Aneurysms in a Case of Arteriosclerosis, Arch. Neurol. & Psychiat. **5**:270-282 (March) 1921.

as well as atheromatous nodules, has been mistaken for miliary aneurysm, and actually it is not so common as originally supposed.

As far as the etiologic factors in the production of the larger aneurysms are concerned, arteriosclerotic or atheromatous degeneration of the media might be considered first as one of the more common causes. These cases of arteriosclerosis and hypertension may be easily recognized from the general examination of the palpable vessels, the heart and the eyegrounds. The medial wall of the blood vessel undergoes fatty or fibrous degeneration, and the wall bulges to form an aneurysm. Aneurysms of this type are more common in the cerebral vessels than elsewhere in the body. In quite a large group of cases, however, there are no evidences of arteriosclerosis anywhere in the body. Moreover, the patients may be quite young. Cardiac hypertrophy, hypertension and peripheral arteriosclerosis may be absent, and the only place where vascular disease appears would seem to be at the site of the aneurysm. Accordingly, some other factor must be present.

Fearnsides, following Eppinger's [11] lead, drew attention to the existence in certain patients of an inherent weakness of the wall of the vessel at a particular place, especially at the points of juncture with other arteries to form the circle of Willis and at the bifurcations. Moreover, microscopic study of the vessels at these points in certain cases may show weakness in structure and abnormalities in development even in persons who have not developed aneurysms during life. This possibly explains the frequency with which aneurysms are found near the circle of Willis. The proportion of relatively young patients who have died from cerebral aneurysm is surprising; of thirty-one patients of Fearnsides, twenty-one were between the ages of 19 and 29. Further, the same author has found record of cerebral aneurysms being observed at the juncture of the left anterior cerebral and anterior communicating arteries in a child, aged 19 months, who had died of some other disease. In two of the cases reported in this article, the patients were 19 and 25, respectively, and each had stigmas of malformation suggesting congenital weakness of the vascular system.

The third factor in the production of cerebral aneurysms, according to Fearnsides' classification, is general infection, vegetative endocarditis and septic emboli. Thirteen of his forty-four cases were in this group. These septic emboli lodge in a cerebral vessel and, besides partly obliterating it, they cause an inflammatory softening of the wall of the vessel so that it bulges and an aneurysm is produced. These may be multiple throughout the body without any signs of the embolus producing the

11. Eppinger, Hans: Pathogenesis (Histogenesis und Aetiologie) der Aneurysmen einschliesslich des Aneurysma equi verminosum, Arch. f. klin. Chir. **35**:1-563 (Supp.), 1887.

arteritis being found. It has been thought that organisms could enter the vasa vasorum from the blood stream and produce arteritis without the intervention of a definite endocarditic embolus. This is much disputed and usually, in aneurysms due to septic emboli, there is a definite endocarditis present, with loose, friable vegetation on the valves of the heart. Accordingly, the three main types of aneurysm, classified according to cause, are the inflammatory, the degenerative, and that due to congenital vascular weakness.

Syphilis seems to play a very small part in the formation of intracranial aneurysm. Fearnsides reported that 90 per cent of aortic aneurysms and 100 per cent of aneurysms of the other large elastic arteries are due to syphilis, yet only 6.25 per cent of the aneurysms of the small muscular arteries can be proved to be due to syphilis; it is only to this class of blood vessel that the cerebral arteries belong. These smaller muscular cerebral vessels suffer an obliterative endarteritis. Instead of a thinning of the wall of the vessel there is a thickening, and instead of dilatation of the vessel there is thrombosis and stoppage of the circulation. Possibly the exception to this rule is in the pathologic change that the basilar and vertebral arteries undergo in the presence of syphilis. In his forty-four cases, Fearnsides could discover no instance in which syphilis seemed to be the sole causal factor, but then in only two of his cases did the aneurysm affect branches of the basilar and vertebral arteries. Other authors have reported many instances of the association of syphilis with aneurysm of the basilar artery, and Fearnsides suggests that the relatively large size of the artery is responsible for its susceptibility to syphilis by dilatation and the formation of aneurysm rather than by obliteration of its lumen.

Trauma is not usually a cause of the type of cerebral aneurysm under discussion although injury may provoke rupture of the distended sac. Arteriovenous aneurysm, formed by communication between the intracranial portion of the internal carotid and the cavernous sinus, is usually due to fracture of the base of the skull and tearing of the artery, so that a communication is established. In the series of 544 cases collected by Locke,[12] about 77 per cent of arteriovenous aneurysms in the cranium were traumatic in origin, while the remainder came on spontaneously. The usual clinical syndrome of this condition, and one easily recognized, is represented by a pulsating exophthalmos, edema of the lids, complete or partial opthalmoplegia on the side affected and a loud systolic murmur audible over the temporal region or even over the whole cranium.

12. Locke, C. E., Jr.: Intracranial Arteriovenous Aneurysm or Pulsating Exopthalmos, Ann. Surg. **80**:1-24 (July) 1924; 272-285 (Aug.) 1924.

CLINICAL SIGNS AND SYMPTOMS

The clinical signs will vary with: (1) the underlying disease responsible for the formation of the aneurysm; (2) the condition of the aneurysmal wall and whether rupture or leakage has taken place, and (3) the site of the aneurysm in the cranial cavity.

Diseases Associated with Cerebral Aneurysm.—In embolic aneurysm, the general or local infection responsible for the production of the aneurysm will dominate the picture. These patients are usually extremely ill, with high fever, chills, delirium, emaciation and general prostration. Commonly there are signs of endocarditis; possibly bacteremia is present, and the patient may die in coma with the aneurysm unruptured and its presence unsuspected; or there may be multiple aneurysms, and the rupture of one of these in the abdominal cavity may cause death before the cerebral aneurysm has ruptured. Death may also come unexpectedly during a period of remission when the patient is up and around. Usually, in cases of sudden hemiplegia occurring during the course of acute septic endocarditis, partial recovery takes place. Later, and unexpectedly, the patient suddenly dies from obvious cerebral disease, and a ruptured embolic aneurysm is found at necropsy. In contrast to the nonembolic types, these aneurysms due to septic embolus are on the branches of the vessels rather than at their origin at the circle of Willis. They are therefore more common on the cortex and in the brain substance than at the base of the cranium. Also and in accordance with these sites, hemiplegia, aphasia and various alternate forms of paralysis are more common in this type. The middle cerebral artery or its branches is supposed to be the most frequently affected, about equally on the right and left side.

ILLUSTRATIVE CASE

CASE 1.—A boy, aged 20, was brought to the Mayo Clinic, March 6, 1924, by his relatives because of his mental condition. There was a history of rheumatic fever at the age of 13. Following this, he had apparently enjoyed good health up to four months before registration, when he began to lose weight and strength. A month later, he had experienced a sudden loss of power in his right lower limb. The limb, however, seemed to recover in a few minutes except for persistent slight limp. One month later, also more or less suddenly, he had had difficulty in expressing himself, but had improved within eight or nine days so that only occasionally he had difficulty in finding the word he desired. Three weeks before registration, he had appeared more weak and listless than heretofore and had complained of pain in the right lower limb and right side of the face. Four days prior to his arrival at the clinic, he had become mentally confused and restless and had refused food. Restlessness increased until he was maniacal and needed restraint.

The patient was emaciated, very restless, noisy, incoherent and picked at imaginary objects in the air and at the bed clothes. There was a loud systolic murmur heard best at the area of cardiac impulse. The transverse diameter of the heart

was increased. There was a slight trace of weakness of the right leg, and Babinski's sign was found on the right side. Aphasia was not evident, although no adequate tests were possible on account of the mental state. The temperature was normal at the first examination. The leukocytes numbered 10,800, and the spinal fluid contained 5 small lymphocytes and 8 polymorphonuclear leukocytes for each cubic millimeter. Blood cultures showed a green-producing streptococcus. For four weeks, the patient's restless, incoherent mental condition continued almost constantly in noisy delirium; he refused all food and had to be fed with a tube. He was incontinent of urine and feces. The temperature during the two weeks preceding death increased to between 100 and 102, and the pulse varied between 100 and 140. Four weeks after arrival, when apparently in his usual state, he suddenly became unconscious, then comatose and died within a few minutes.

At necropsy were found chronic vegetative mitral and left auricular endocarditis, embolic aneurysm of the left middle cerebral artery with rupture and hemorrhage, multiple infarcts of the spleen and kidneys, hypertrophy of the heart (514 Gm.) with fatty changes and adhesive pericarditis, hypertrophy of the liver (2,672 Gm.) and embolic aneurysm in the pancreas.

The clot surrounding the ruptured aneurysm was brownish red at the periphery but green in the center, and measured 6 by 4 cm. It was at the base of the brain in the middle cranial fossa and partly in the sylvian fissure. The aneurysm in the pancreas was a sacculated aneurysmal dilatation of the superior pancreaticoduodenal artery, measuring 4.5 by 3.5 by 3 cm. It was filled with a large laminated thrombus.

Comment.—There had evidently been a recrudescence of the original chronic vegetative endocarditis with dispersion of emboli throughout the body. The beginning of the formation of the aneurysm was signaled by the appearance of a right monoplegia four months before death. This was hardly noticeable at the time the patient was under observation at the clinic, and his persistent delirium was the outstanding feature for the four weeks prior to death. The delirium was partly due to the infection and toxemia, but probably the aneurysm also played a part. The endocarditis, however, dominated the clinical picture and obscured the development of a mycotic aneurysm of the left middle cerebral artery. However, considering all other necropsy data, including the large unruptured aneurysm in the pancreas, the cerebral aneurysm represented only a part of the whole pathologic picture.

In arteriosclerosis and hypertension, the age of the patient, the blood pressure and the cardiac hypertrophy, together with manifest degenerative signs in the peripheral and retinal vessels, are enough to suggest the underlying cause of the aneurysm. There will also be present the usual symptoms of cerebral vascular disease in the form of vertigo, headaches, deafness, tinnitus and failing memory. Syphilis is usually detected by the serologic examination of the blood and spinal fluid as well as by the traces of previous infection revealed by the general physical examination. In cases in which there is congenital weakness of the walls of the vessel, there may be no other signs or symptoms manifest than those produced by the aneurysm. It is possible, however, to find in some

instances stigmas of faulty development of the vascular system, as is well shown in cases 2 and 3. More often, nothing is found in the general examination to indicate the reason for the development of an aneurysm; it is for this reason that the diagnosis of these cases is usually so difficult. The age of the patient at the onset of symptoms also varies with the underlying cause of the aneurysmal dilatation. Embolic aneurysms and those due to congenital weakness of the wall of the vessel occur roughly within the first four decades of life; the aneurysms due to arteriosclerosis and hypertension appear later.

CONDITION OF THE ANEURYSMAL SAC IN RELATION TO SIGNS AND SYMPTOMS

Next to the causal disease, the varying state of integrity of the wall of the aneurysm tends to modify the course of events. Widely different clinical syndromes appear in aneurysms: (1) that have remained unruptured during life; (2) that have ruptured completely with free hemorrhage into the subarachnoid space with subsequent death of the patient, and (3) that have leaked intermittently prior to the final and lethal rupture.

Aneurysms that do not rupture are the least likely to give rise to signs during life; it is more usual for them to be discovered accidentally at necropsy following death from some other disease. Thus, 16.5 per cent of Beadles' 555 patients and 16.1 per cent of Fearnsides' patients died without sign or symptom of aneurysm having been evident during life. Not all unruptured aneurysms fail to produce symptoms; when they grow large enough to compress surrounding structures and produce indentation anemia and softening, signs of their presence are bound to occur. These will of course depend on the structures affected, and the signs are usually of focal compression rather than those of generally increased intracranial pressure. In only one of Fearnsides' thirty-one cases were there symptoms prior to rupture, and these were vague; but in 16.4 per cent of Beadles' 555 cases there were symptoms of intracranial tumor prior to the fatal seizure, and in 20.7 per cent "signs more or less suggestive of tumor" were present. The literature contains enough reports of individual cases of aneurysm producing signs of focal compression and, in at least two instances, exploratory craniotomy was performed under the erroneous supposition that there was a neoplasm near the optic chiasm or cavernous sinus. On the whole, however, it seems that aneurysms in the region of the circle of Willis seldom reach a large size before they leak or rupture. Other signs are then usually present besides those of focal compression. As a contrast, there are many recorded cases in the literature of aneurysm of the basilar and vertebral artery having attained a size sufficient to cause compression of

the pons or medulla with corresponding symptoms before death by rupture or intercurrent disease.

While the symptoms of aneurysm before rupture are mainly those of any expanding body in the cranial cavity, a very different picture is produced when rupture ensues. Rupture or leakage of a cerebral aneurysm is the more common termination of events. In Beadles' series rupture had taken place in 339 (61 per cent), and in Fearnsides' cases of embolic aneurysm this accident had occurred in twenty-five (80.7 per cent). The rupture may be complete and final and death ensue within a few hours or days. The onset of symptoms is usually sudden, like an attack of apoplexy, and there is little evidence of localization present beyond that of extensive basal cerebral hemorrhage. Consciousness is lost immediately or within a short period, and coma ensues; the pupils are widely dilated, the color cyanotic, the breathing stertorous and the pulse at first slow and later rapid and weak. Convulsions may result from cortical irritation; occasionally hemiplegia or ocular paralysis appears, and the neck may be rigid from irritation of the occipitocervical meninges by extravasated blood. The spinal fluid obtained by lumbar puncture is bloody. Two of Fearnsides' patients died before they could be admitted to the hospital, having been picked up dead near the spot where the fatal rupture took place. Usually in complete rupture of the sac there are few signs to indicate the site of the rupture, and death is the rapid outcome of the widespread shock to the intracranial contents.

Much more interesting clinically are the cases of intermittent leakage of blood from the aneurysmal sac with alternating episodes of disturbed function and periods of relatively good health. This phenomenon occurred in fifty-seven (31.1 per cent) of Wichern's[13] cases, and in Fearnsides' series it was present in 41.9 per cent. In these cases there was a history of two, three or more apoplectiform seizures separated by varying intervals. Sooner or later an episode culminated in death. At necropsy the pathologic counterpart of these episodes was found in different layers of old and recent hemorrhage from a ruptured aneurysm at the base of the brain. This general behavior is characteristic and diagnostic in a large percentage of cases of intracranial aneurysm and makes it possible to recognize the disease during life. The periods separating individual attacks may be quite long; in one of Fearnsides' cases no less than twenty years intervened between two seizures. One of his patients had had five attacks before death; the first attack was twenty-seven days before, the second five, the third three, the fourth two days, and the fifth a few minutes before death. The onset of these attacks is usually sudden and associated with a sudden severe headache or sensation of something giving way inside the cranial cavity. They may follow sudden exertion.

13. Wichern, Heinrich: Zur Diagnose perforierender Aneurysmen der Hirnarterien, München. med. Wchnschr. **58**:2724-2729, 1911.

Case 2.—A boy, aged 20, was brought to the Mayo Clinic, May 2, 1925, in a state of deep stupor. He had always been active and athletic and apparently well up to five weeks before. At that time, after having had to push a motorcycle over a mile of sandy road, he fell suddenly for no apparent reason. He was not unconscious and arose immediately and was able to ride his motorcycle home. Following this he seemed as well as usual but complained of a feeling of pressure in the head. Two weeks before admission, while playing baseball, he suddenly experienced a severe pain in the occiput, became dizzy and staggered home, was put to bed, vomited several times and complained of intolerance to light. He was restless all that night but was better in the morning and was kept in bed because of headache and photophobia. He seemed to improve during the succeeding twelve days but remained in bed. The evening before being brought to the clinic, he went to sleep at the usual hour but awoke near midnight screaming because of a sudden severe pain in the occipitocervical region. He cried out that his neck was broken and that he was going to die. He became unconscious within a few hours and remained so up to the time that he was first examined.

The patient was well nourished; he was unconscious but restless, with a pulse rate of 60 and a temperature of 101 F. The neck was rigid, and both Kernig and Babinski signs were present bilaterally. The eyegrounds were full of large hemorrhagic areas around the optic disks, and there was a mild edema of the nerve heads. The spinal fluid was bloody, containing almost pure blood under pressure. A harsh systolic murmur was heard more easily over the aortic area, and an easily palpable thrill was felt over the area of the aorta and carotid vessels. Examination of the blood vessels showed some curious anomalies: the right carotid artery crossed the trachea in the region of the jugular notch; compression of the left carotid artery caused the left radial pulse to disappear; no pulsation was felt in the left subclavian artery; a small vessel descended from the mastoid region into the posterior cervical triangle to reach the outer end of the left clavicle, compression of which also diminished the radial pulse; large tortuous pulsating vessels could be felt over the back between the scapulae, and in the lateral aspect of the thorax in the postaxillary line near the inferior angle of the scapula; no pulsation could be felt in the abdominal aorta, and only slight pulsation in the right femoral artery, but in no other vessel in the lower extremities could the pulse be felt. The blood pressure in the right arm was 164 systolic and 86 diastolic; in the left arm it was 126 systolic and 110 diastolic. The attempt to measure the pressure in the lower extremities showed only a few small oscillations between 80 and 90 mm. of mercury.

The patient lived only eleven days, during most of which time he was unconscious. On one occasion he roused sufficiently to talk naturally, but only for a few minutes. The papilledema increased. He developed weakness of the right side toward the end, and he had one general convulsion. Repeated spinal puncture showed bloody fluid. The temperature fluctuated between 101 and 103 F.; on two occasions it went down to normal. The pulse rate steadily rose from 50 to 180 and toward the end Cheyne-Stokes' respiration appeared, and he died in coma.

Necropsy showed congenital atresia of the isthmus of the aorta with an extensive collateral circulation. The right internal carotid artery was larger than the left. The right vertebral artery in the cranium was quite large, while the left was smaller than normal. There were two aneurysms on the circle of Willis. One was a dumb-bell-shaped dilatation of the left posterior communicating artery at its juncture with the middle cerebral, which had ruptured. The other was situated on the anterior communicating artery at its juncture on the right side with the anterior cerebral artery.

Comment.—This patient had a rare and interesting disease wherein there had been congenital closure of the aorta at the origin of the fetal ductus arteriosis. There was only a fibrous cord to represent the left subclavian artery. An extensive collateral circulation had been developed to replace the obliterated vessels. The development of the aneurysms at the circle of Willis was probably due to congenital weakness of the vessels at their points of juncture, as already discussed, and is in accord with the anomalous and faulty development elsewhere in the vascular system. It is also possible that there was a mechanical factor in the unequal blood pressure on the two sides of the arterial tree. An abnormally high systolic pressure is likely to be developed to drive the blood through the tortuous ramification of the collateral circulation, and the pressure in the right upper extremity and probably also in the head and neck was much greater than in the left upper extremity, trunk and limbs, where it was extremely low. All the circulating blood for the body had to be diverted through the vessels supplying the head, neck and upper extremities. Records of cases of the same disease show instances of a large aneurysm of the right subclavian artery due to the strain of bearing a high blood pressure concentrated on the right side of the head and neck.[14]

The initial leakage of blood into the cranial cavity followed the undue exertion the patient went through in pushing his motorcycle through a mile of sandy road. This probably elevated the blood pressure considerably and, by throwing a great strain on the vessels of the right arm, head and neck, caused the aneurysm to rupture. The intermittence of symptoms due to repeated leakages from the aneurysm is apparent. Leakage had occurred at least three times. That just prior to the admission of the patient to the clinic was a severe one, and the general clinical picture while he was under observation was one of a profuse hemorrhage at the base of the brain without any definite localizing signs.

The large hemorrhagic areas that were found in the fundus of the eye are occasionally seen in cases of ruptured cerebral aneurysm. Doubler and Marlow [15] have already described a case in which this condition was observed before death, and the dural sheath of the optic nerve was filled with blood at necropsy. Apparently the blood is forced into this sheath and then through the lamina cribrosa to appear as large, blotchy hemorrhagic areas on ophthalmoscopic examination.

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14. Woltman, H. B., and Shelden, W. D.: Personal communication to the author.

15. Doubler, F. H., and Marlow, S. B.: A Case of Hemorrhage into the Optic Nerve Sheaths as a Direct Extension from a Diffuse Intrameningeal Hemorrhage Caused by Rupture of Aneurysm of a Cerebral Artery, Arch. Ophth. **46**:533-536 (Nov.) 1917.

Case 3.—A married man, aged 52, was brought to the clinic, Feb. 14, 1923. His brother, who accompanied him, stated that the patient had apparently not been acutely ill when they started out two days before. He had, however, complained of headache on the train, had become drowsy, and during the last four hours of the journey had been in a stupor. According to information variously obtained, the patient had been apparently well up to three years before. At that time he had experienced an attack of right lumbar pain radiating to the groin and testis on the same side. This had lasted a few hours and was relieved by a hypodermic injection. His local physician had diagnosed this as an attack of renal colic. Since then there had been occasional milder attacks of unknown number, but the clinical and laboratory observations at the time of the attack were unknown. For four months he had been complaining of quite constant, mild epigastric and right hypochondriac pain, not dependent on diet or any physical function. He had lost 10 pounds (4.5 Kg.) and also considerable strength. Four months before admission, while working in the fields, he had complained of sudden, severe frontal headache; having become dizzy, he had attempted to walk home, but had fallen unconscious in the yard. Within thirty minutes, he had shown signs of returning consciousness; he had vomited and had been irrational or profoundly lethargic alternately for three days before regaining full consciousness. The temperature had been slightly elevated for five days, and he complained of double vision. Within three weeks he recovered sufficiently to be up and around. He was clear mentally but complained of headache, and had not been able to work from that time on. The second attack took place two months later. It also came on suddenly with vertigo, frontal headache and unconsciousness, all appearing within the first five minutes. He was stuporous, this time for only two days, and gradually recovered mentally and physically after nine days in bed. For the remaining six weeks before visiting the clinic he was up and around, and complained only of general weakness, mild abdominal pain and occasional frontal headache. He visited his brother in another part of the state before admission and at that time seemed well except for abdominal pain.

The patient was completely unconscious on admission. His temperature was normal, but in the succeeding five days it rose gradually and was 102 at death. His pulse was 80 on arrival and varied between that and 60 until the last day, when it rose suddenly to 140. The blood pressure was 100 systolic and 70 diastolic; there was some tenderness over the abdomen at the umbilicus, of which the patient was aware during his lucid intervals.

He lay quietly in bed with his head somewhat retracted, and occasional irregular twitchings were observed in all four extremities. The neck was extremely rigid, and both Kernig's and Lasègue's signs were present. Examination of the urine was negative. The blood urea was normal in amount. There was a moderate leukocytosis of 13,000, 10,500 and 11,250 on three counts. The Wassermann reactions of the blood and spinal fluid were negative. The spinal fluid, however, was cherry red, opaque and contained fresh blood just as long as the fluid was allowed to run. The pressure was definitely increased. No abnormal elements except the blood were discovered in the fluid.

Ophthalmoscopic examination showed nothing unusual in the eyegrounds. The pupils were equal, regular and reacted promptly to light. Roentgen-ray examination of the head and chest was negative from the standpoint of tumor or metastasis. There were very few neurologic signs beyond those of meningeal irritation. On the third or fourth day a slight ptosis of the left upper eyelid and slight flattening of the left side of the face appeared. There was a tendency to keep the arms flexed at the elbows with the wrists tucked under the chin. During the five days

prior to death, there was little change except in the degree of mental clouding. The extreme rigidity of the neck remained constant, as did the Kernig sign, but toward the fourth day the patient became somewhat brighter mentally and could be aroused for short periods only to relapse if stimulation ceased. He was able to recite some of his history and complained of severe supra-orbital bilateral headache and of pain when he attempted to flex his neck. The morning of the fifth day, he suddenly became rigid all over and developed stertorous breathing, cyanosis and slow respiration. After a few minutes he relaxed, but from then on respirations became slower and slower, and the pulse increased in rate and became irregular. He died on the morning of the fifth day after admission.

At necropsy, aneurysm of the right middle cerebral artery with multiple ruptures and basilar hemorrhages, carcinoma of the left suprarenal with invasion of the left renal pelvis, and arteriosclerosis of the aorta and cerebral arteries were found. There were no metastatic lesions in the lungs, liver or elsewhere in the body. Microscopic sections of the cerebral vessels showed no neoplastic tissue, but definite arteriosclerotic degeneration of the middle coat with calcification was present in a number of the cerebral vessels.

Comment.—The clinical picture presented by this patient was one of irritation of the meninges at the base of the brain. It was somewhat similar to that in case 2, but there were no evident congenital anomalies of the vessels, and the apparent cause was arteriosclerosis. The stupor, profound rigidity of the neck and muscular irritability suggested meningitis, but the temperature was not much elevated and the first puncture showed bloody fluid. The clinical picture was quite characteristic of that recently described by Packard and Zabriskie.[16] The history revealed clearly the intermittence of attacks and the characteristic episodic apoplectiform attacks already mentioned. From all accounts obtained, very little was to be noted in the interim between the three apoplectic seizures. The patient was even able to make a railroad journey, and not even his brother had noted any change in his condition between the second and last seizures. The association of hypernephroma with this disease has been recorded in one other case described by Rhein.[17] Whether the association was merely accidental or was of some fundamental importance, it is hard to say. The apparent disease mainly responsible for the formation of the aneurysm in the present case was the arteriosclerosis evident in the cerebral vessels (fig. 1).

INFLUENCE OF THE SITE OF THE ANEURYSM

The more specific neurologic signs indicative of the site of these intermittent leakages vary according to the particular vessel involved. As has been mentioned, the region of the circle of Willis, at the bifurca-

16. Packard, Maurice; and Zabriskie, E. G.: Basal Cerebral Hemorrhages, J. A. M. A. **85**:1633-1637 (Nov. 21) 1925.

17. Rhein, H. W.: Aneurysms of the Vessels of the Brain, J. Nerv. & Ment. Dis. **41**:360-367, 1914.

tion of its component vessels or at their junctures, is the usual point for aneurysm to develop. According to Gowers,[18] in 154 cases of cerebral aneurysm the middle cerebral artery was most often involved, being affected in forty-four cases. The basilar artery was involved in forty-one cases; the internal carotid in twenty-three, the anterior cerebral in fourteen, the posterior communicating in fourteen, the anterior communicat-

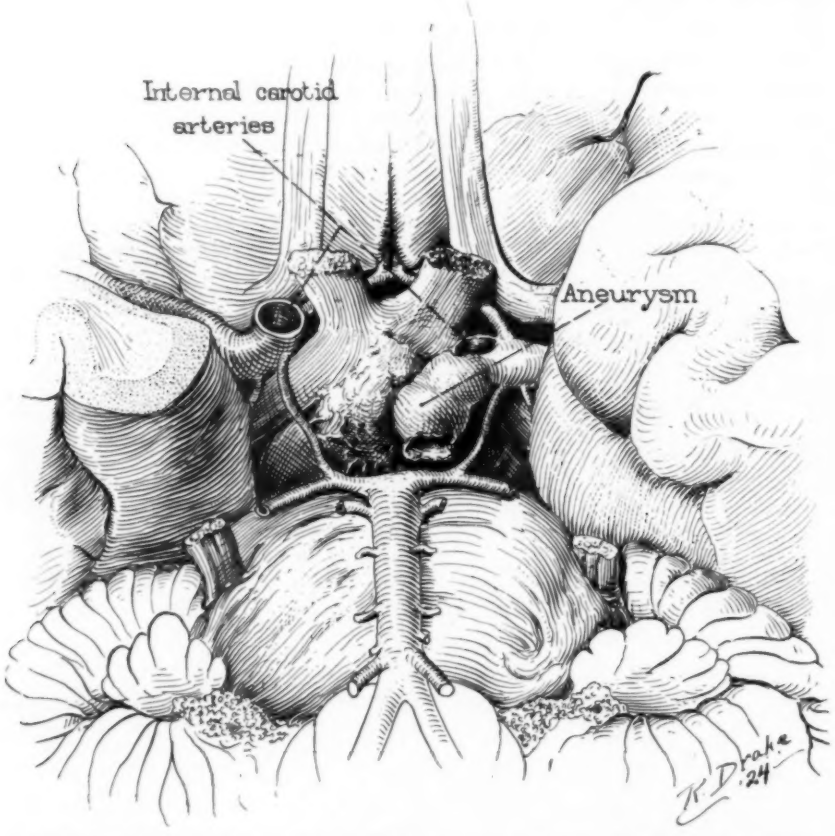

Fig. 1.—Aneurysm of the right middle cerebral artery with multiple ruptures and basilar hemorrhage.

ing in eight, the vertebral in seven, the posterior cerebral in six and the inferior cerebellar in three. The middle cerebral and basilar arteries, according to these data, seem by far the most commonly affected. The signs and symptoms corresponding to these individual blood vessels can for convenience be grouped according to this attachment of vessels to the anterior or posterior portion of the circle of Willis. The anterior

18. Gowers, W. R.: A Manual of Diseases of the Nervous System, ed. 2, Philadelphia, P. Blakiston's Son & Co., **2**:529, 1893.

group comprises the internal carotid, middle cerebral, anterior cerebral and anterior communicating arteries; the posterior group comprises the vertebral arteries and the basilar and posterior communicating arteries.

The Anterior Group.—There is a rather definite syndrome representing aneurysms of the internal carotid and middle cerebral arteries depending on compression or on leakage. Those due to pressure on contiguous structures, as in the case of any expanding body in the vicinity of the cavernous sinus and medial wall of the middle cranial fossa, are: pain in the first or ophthalmic divisional area of the trigeminus; paralysis of the third, fourth and sixth cranial nerves; exophthalmos; occasionally papillo-edema; and rarely, as in the cases of Bramwell [19] and Mitchell,[20] erosion of the sella with dyspituitarism, optic atrophy and hemianopia. The pain over the forehead and supraorbital ridge is severe and neuralgic, and there may be hypesthesia in that area with loss of the corneal reflex. Symonds' [3] case showed this syndrome, and in Shannon's [21] case there was a central scotoma with pallor of the optic disk on the side of the aneurysm and mild signs of disturbance of the pyramidal tract on the opposite side. The other signs present are due to leakage from the aneurysm from time to time; the signs of focal compression are suddenly intensified, and there is evidence of extravasation of blood at the base of the brain. The spinal fluid, at first bloody, later becomes yellow in the intervals between the attacks. There may be disturbance of consciousness in these attacks, varying from mild confusion to delirium or stupor. There is usually a mild pyrexia of between 99 and 101 F. during the attack, and the clinical picture may sometimes closely resemble acute meningitis and might be mistaken for it were it not for the free blood in the spinal fluid and the suddenness of the onset. This picture of meningeal irritation from extravasated blood may occur in all cases of aneurysm even when the lesion is as far forward as the anterior cerebral artery. When the irritation is severe, it masks all localizing signs. The signs of aneurysm of the anterior cerebral artery or the anterior communicating artery, without rupture or with only mild leakage, resemble those already described in connection with aneurysm of the middle cerebral and internal carotid, except that the lesion being rather far forward, bitemporal or

19. Bramwell, Byrom: Clinical Lecture on Intracranial Aneurysms and Meningeal or Extracerebral Hemorrhage, Clin. Studies, 1905-1906, pp. 289-325.

20. Mitchell, S. W.: Aneurysm of an Anomalous Artery Causing Anteroposterior Division of the Chiasm of the Optic Nerves and Producing Bitemporal Hemianopsia, J. Nerv. & Ment. Dis. **16**:44-62, 1889.

21. Shannon, J. R.: A Case of Aneurysm of the Internal Carotid (Intracranial Portion) and Its Effect on the Patient's Vision, Arch. Ophth. **46**:518 (Nov.) 1917.

homonymous hemianopia and loss of smell are more likely to be produced from involvement of the optic chiasm and olfactory nerves.

CASE 4.—A man, aged 21, came to the Mayo Clinic, Sept. 13, 1925, complaining of double vision and numbness of the right side of the face. Two years before, while wrestling, he was thrown on his head and knocked unconscious for fifteen minutes, but recovered rapidly and seemed to have no residual trouble. Fourteen months before admission, he suddenly developed double vision which was more marked on looking to the right. This had remained unchanged up to the time of his visit to the clinic. Three weeks before admission he had suffered from severe bilateral frontal headaches for two or three days, and thereafter had noticed that the right side of the face was numb. This numbness was followed in a few days by difficulty in chewing on the right side, and he felt that his jaws had become weak on that side. For the three or four days before he arrived, he had had attacks of sudden vomiting without headache or nausea. His voice had become higher in pitch for the week previous.

The patient was well developed and in good physical condition except for his presenting complaints. There was apparently a congenitally anomalous development of the whole left side of his body. The arm and leg on that side were larger than on the right, and the skin over the left side of the neck and thorax, the left arm and shoulder and left buttock was cyanotic with enlarged venules. It was thin and easily picked up between the finger and thumb. There was a lack of subcutaneous fat over this entire area and the skin lay in folds. The radial vessels on this side were thickened and sclerotic, but the blood pressure was normal and equal on the two sides. There was a pedunculated tumor, apparently lipoma, hanging from the skin over the left pectoral muscle. The right sixth cranial nerve was completely paralyzed, and the area on the right side of the face supplied by the fifth nerve was markedly hypesthetic to pain, touch and temperature. The masseter, temporal and pterygoid muscles on this side were almost completely paralyzed. The eyegrounds were normal. There were no changes in the tendon reflexes, but Oppenheim's and Gordon's signs were positive on the left side. The spinal fluid was yellow, clear and under a pressure of 10 cm. of water. The Wassermann test of the blood and the spinal fluid was negative.

For the first week, before admission to the hospital, the patient was up and around, although signs progressed to the point that complete paralysis of the right third and fourth cranial nerves was manifested, with the paralysis of the sixth already present. Paralysis of the right seventh cranial nerve also appeared, but it was mild at first. On the seventh day, he became rather suddenly worse. He complained of severe headache and was somewhat drowsy and difficult to arouse. The neck was rigid, and Babinski's sign was present bilaterally. The third, fourth, fifth and sixth cranial nerves on the right were paralyzed, and the third and fourth cranial nerves on the left were affected. He became stuporous and failed to respond to stimulation. A second spinal fluid examination showed a deep yellow, cloudy fluid containing fresh blood cells. It was under a water pressure of 20 cm. Following this drainage of spinal fluid, he improved again and seemed more alert and more responsive. The ophthalmoplegia remained the same. He continued about the same for six days. He was drowsy but could be aroused, slept most of the time but answered questions and ate his meals. Two more examinations of the spinal fluid showed that it was pale yellow and contained many red blood cells but apparently no fresh blood. The pressure had, however, risen to 30 cm. of water. The rigidity of the neck was not marked, and he seemed to be holding his own as far as the disease was concerned. The end,

however, came suddenly on the thirteenth day when he became stuporous, had two tonic convulsions with stertorous breathing and died within two hours of the onset of the stupor.

The necropsy showed a saccular aneurysm of the right internal carotid artery in its course in the cavernous sinus. This aneurysm had ruptured into the right temporal lobe of the brain and into the ventricular system. There had been pressure on the right third, fourth, fifth, sixth and seventh cranial nerves and the left third and fourth nerves. There was also a fusiform aneurysm of the left internal carotid artery in the carotid canal. The circle of Willis showed some anomalies; the left vertebral artery was larger than the right, and there was a large communicating vessel between the right posterior inferior cerebellar artery and the anterior inferior cerebellar artery. About 3 mm. distal to the juncture of the two vertebral arteries, there was a fusiform dilatation of the basilar artery about 4 mm. in length. The distal end of the basilar divided into two branches, which ran directly to join the internal carotid without the intervention of a posterior communicating branch. The posterior cerebral arteries arose from this continuation of the basilar.

Comment.—In this case there was considerable evidence of congenital, vascular maldevelopment.. The large nevus that was found at the first examination was enough to suggest other congenital anomaly in the vascular system. The course of the disease was characteristic and could be interpreted either as a progressive enlargement of the aneurysm or more likely as a series of hemorrhages. The localization of the aneurysm here was definite. With progressive involvement of the third, fourth and sixth nerves and the anesthesia over the right side of the face, a lesion near the cavernous sinus was immediately thought of. The first spinal fluid was yellow and not under pressure, which indicated that hemorrhage had occurred previous to that date. The second spinal fluid, however, showed red blood cells and corresponded to a sudden change in the patient's condition for the worse. This evidently indicated a fresh hemorrhage with the development of the final stage of the disease (fig. 2).

The Posterior Group.—Excluding instances of sudden and final rupture before any signs become manifest, the clinical picture produced by aneurysm of the basilar and vertebral arteries is very different from that already described for vessels lying further forward. These aneurysms in the posterior cranial fossa tend to reach a relatively large size before they rupture. Accordingly, signs of pressure on contiguous structures uncomplicated by signs of leakage are more frequent. The basilar artery is usually involved in its superior part and near its bifurcation. Krabbe and Backer [22] recently collected seven cases from the literature and described one of their own. The disease had the same tendency to proceed by remissions and exacerbations as elsewhere. The characteristic

22. Krabbe, K. H., and Backer, K. H.: Contributions au diagnostic des anévrismes de l'artère basilaire du cerveau, Acta med. Scandinav. **56**:95 (March 22) 1922.

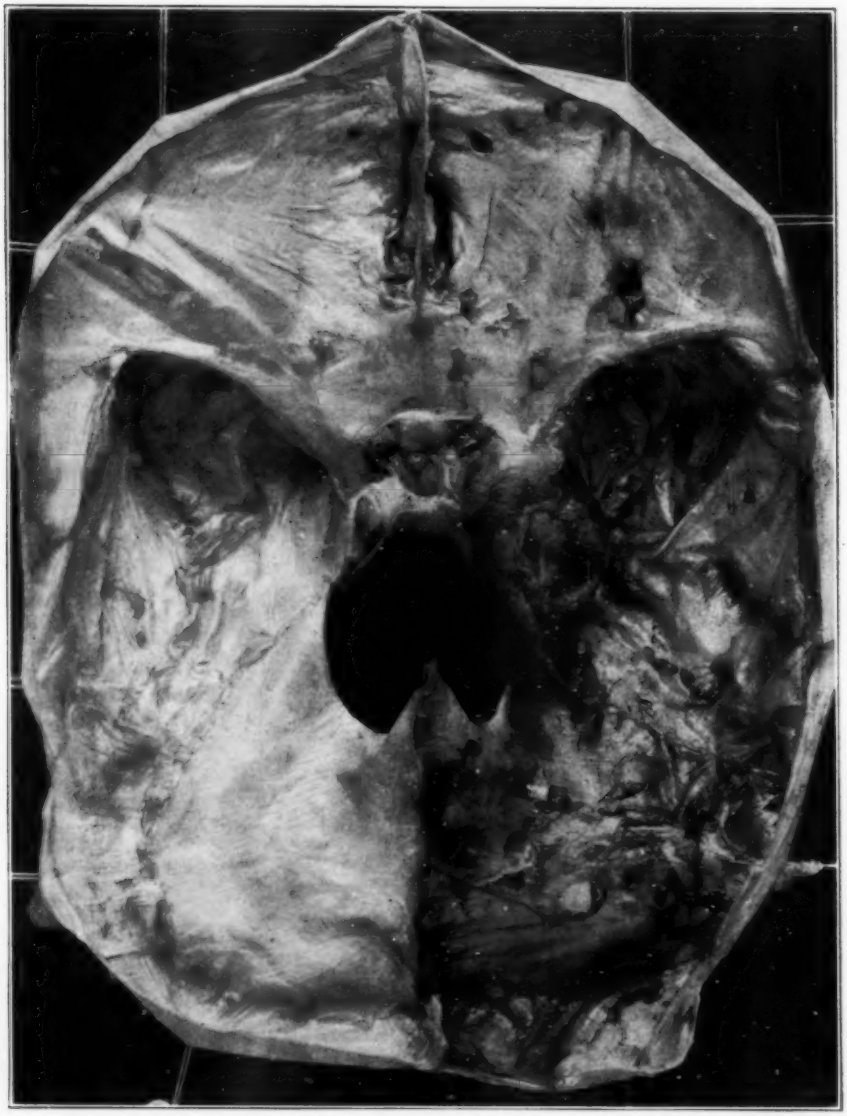

Fig. 2.—Saccular aneurysm of the right internal carotid artery with rupture into the temporal lobe of the brain and middle cranial fossa.

feature of aneurysm of the basilar artery is the sudden onset of hemiplegia that disappears and reappears later on the opposite side in intermittent attacks; sometimes it alternates with paraplegia without disturbance of consciousness. Right-sided hemiplegia without aphasia may appear suddenly, last some weeks, slowly improve, then within some months paraplegia or fresh hemiplegia may appear on the left side. Aneurysm of the basilar artery only can cause paraplegia. Dysarthria, dysphagia and mild cerebellar signs are usually present and sometimes alternate with paralysis of the seventh cranial nerve. The aneurysm grows slowly, and when rupture occurs there are the same signs as in intracranial aneurysm generally, only there is a more sharply localized occipital headache and a tendency to pull the neck over to one side.

In case of aneurysm of the vertebral artery the left is more usually affected because it is likely to have a constriction where it joins the right artery to form the basilar. These aneurysms are also likely to be large, as in Well's [23] case in which it was 30 mm. in diameter and 35 mm. in length. Here also the course is intermittent; in Monakow and Ladame's [24] case there was a series of violent attacks of vertigo with unilateral deafness and tinnitus ending in permanent ataxia of gait. In this case heterolateral hemianesthesia, dysphagia, dysarthria and irregular pulse rate with dyspnea were also present. Toward the end, delirium ensued, and the patient died from paralysis of the respiratory centers. In Well's case also, paroxysmal vertigo and tinnitus were manifested together with syncopal attacks and pain in the jaws. Apparently, therefore, the eighth, ninth and tenth cranial nerves are more frequently affected by this aneurysm.

In Howe's [25] case there was also paralysis of one side of the tongue, face and sternomastoid, but the diagnosis was not proved by necropsy. Oppenheim [26] mentions that in these cases of aneurysm of the posterior cranial fossa there is disturbance of respiration when the head is bent forward on the chest, and that breathing may be arrested if this is persisted in. In cases of basilar as well as of vertebral aneurysm, a systolic pulsating bruit has been described which can be heard by the patient, and by the physician when he auscultates the skull. This may be loudest over the mastoid or occiput. This seems to be very seldom if ever present in cases of aneurysm of the anterior group of vessels; in

23. Wells, H. G.: Intracranial Aneurysm of the Vertebral Artery, Arch. Neurol. & Psychiat. **7**:311 (March) 1922.

24. Monakaw, C. von, and Ladame, P.: Anévrysme de l'artère vertébrale gauche (autopsie), Nouv. iconog. de la Salpêtrière **13**:1-25, 1900.

25. Howe, H. S.: Aneurysm in the Posterior Cranial Fossa, Neurol. Bull. **2**: 323-328 (Sept.) 1919.

26. Oppenheim, Hermann: Textbook of Nervous Diseases, London, T. N. Foulis **2**:934, 1911.

any event, Oppenheim doubts the reliability of this sign because subjective and objective intracranial pulsating bruits can be present in diseases other than aneurysm, such as hydrocephalus, anemia, vascular tumors or even in cases of compression of blood vessels by tumor.

As a rule there are no characteristic signs produced by aneurysm of the posterior communicating and posterior cerebral arteries, except that Oppenheim mentions alternate hemiplegia with paralysis of the third nerve (Weber's syndrome) as a feature in the latter due to pressure on the midbrain.

CONCLUSIONS

1. In four cases at the Mayo Clinic, arteriosclerosis was the cause of aneurysmal dilatation in one, endocarditis with embolism in one and congenital weakness of the arterial wall in two.

2. The signs of embolic aneurysm are likely to be masked by those of the causal disease; that is, endocarditis.

3. Congenital weakness of the wall of a cerebral artery with the formation of aneurysm may be associated with congenital anomaly elsewhere in the vascular system.

4. In cases in which complete and final rupture has taken place, the symptoms are those of hemorrhage at the base of the brain with signs of meningeal irritation. The spinal fluid is bloody.

5. It is only in the cases of aneurysm with intermittent leakage and focal compression that correct diagnosis and localization of the aneurysm is possible.

PSYCHOGALVANIC STUDIES IN SCHIZOPHRENIA*

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One of the most reliable physiologic indicators of emotional reaction is the psychogalvanic reflex. In the work here reported an attempt was made to determine typical features of this phenomenon in a number of psychopathologic reaction types and to find out to what extent its variations can be used for discrimination and characterization of various emotional and personality disorders. Experiments on several groups of normal persons served as a control. Our aim was to expose the subjects to the same set of stimuli, to compare their reactions, and to link these up with the characteristic features of their emotional and behavior picture. A part of the experimental work was done with Dr. C. P. Richter, another part with Dr. E. F. Kinder. The technic followed was developed by Dr. C. P. Richter and has been employed for a number of years in the Psychobiological Laboratory of the Henry Phipps Psychiatric Clinic.

The subject was seated in an arm-chair during the test, his forearms being supported by rests to which the electrodes were attached. These electrodes, made of zinc disks 2½ inches (6.27 cm.) in diameter, were connected with the backs and palms of both hands by a paste of kaolin and saturated zinc sulphate solution. This conductor renders the electrodes nonpolarizable. No external current was introduced into the circuit, and the fluctuations of the galvanometer string resulted entirely, therefore, from an electromotive force originating in the subject. The instrument used was a Hindle string galvanometer of the type employed for electrocardiography, with a camera for recording the deflections on a rolling film paper. The instrument and the experimenters were kept out of sight of the subject by a curtain dividing the room. Since we considered it important to avoid visual and other extraneous stimulation, we dimmed the light in the room and eliminated, as far as possible, stimuli not belonging to the test. In order that the subjects might be in a purely passive and receptive state, we asked them to sit quietly and to make no response during the experiment, since the production of a reaction word might possibly be a factor unnecessarily complicating the experiment and its interpretation.

Before the stimuli were given the galvanometer string was standardized to the same sensitivity for all subjects (the introduction of 1 millivolt causing 10 millimeter deflection of the shadow of the string).

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* From the Psychobiological Laboratory, Henry Phipps Psychiatric Clinic, Johns Hopkins Hospital.

Verbal and other sensory stimuli were presented. We did not follow any of the lists of association words commonly used; instead we put together a collection of about 100 words, indicative of a variety of life situations, such as family, sex, social standing, morals, money, religion, success, self-esteem. The words used thus touch practically all those topics which are likely to arouse an emotional response in normal persons as well as in patients. The possibility that such a list would present too many complex words was not substantiated in our experience; it was evident that an excessive number of reactions was present only in persons of especially sensitive and responsive emotional organization. The additional sensory stimuli used were the sounding of an automobile horn, pin pricks and the threat of a pin prick. A record of the respiratory movements also was taken during the thirty to forty minutes of experimentation.

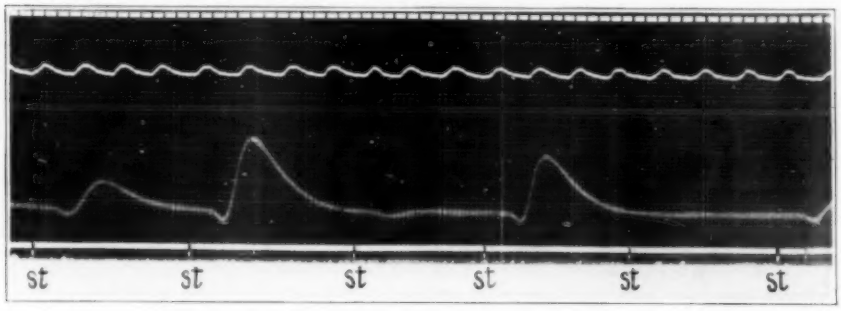

Fig. 1.—Part of film recording the time of stimulation (*st.*), the galvanic curve, the respiratory curve and the time in seconds; the galvanic reactions follow the stimuli directly.

This experimental arrangement, though not ideal, makes possible, nevertheless, a fine registration of emotional characteristics in various persons. Very few of our subjects seemed to be disturbed by the experimental procedure. Whatever the shortcomings of the method may be, they are not significant enough to obscure those features which are typical and consistent for the individuals of the various groups.

Before presenting the results concerning the psychogalvanic reflex, I wish to mention incidentally some outstanding characteristics of the body resistance noted in these studies. The resting current and the body resistance (to a current of 1 milliampere) were measured both at the beginning and at the close of the experiment. The different groups of persons showed typical variation in resistance. Only the more outstanding differences will be mentioned here. The average electrical resistance in catatonic stupor patients was found to be more than twice as high (280,000 ohms) as it is in normal persons (111,000), and in

the former the difference in resistance between the two hands was unusually large. Though the average resistance of paranoid schizophrenic patients (120,000) was similar to that of normal persons, they showed a peculiar lability of resistance, that is, an excessive drop during the test. In a group of fifteen depressions there was rather the opposite tendency; there was relatively little change of resistance during the test, and the average resistance (216,000) was decidedly higher than in normal persons though not as high as in catatonic patients.

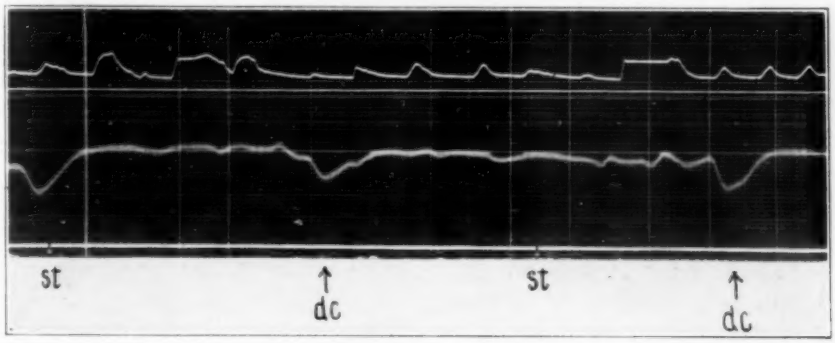

Fig. 2.—Disconnected (*dc*) reactions (occurring later than eight seconds after the respective stimuli); the galvanic and especially the respiratory curves are of irregular shape.

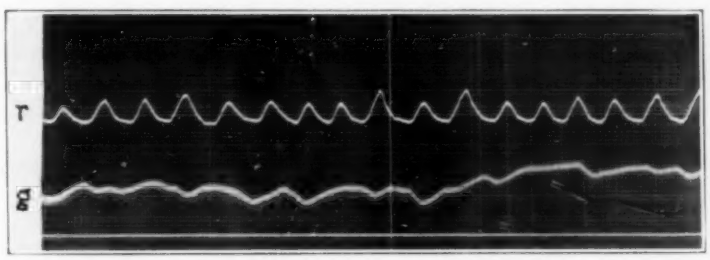

Fig. 3.—Numerous spontaneous waves (no stimuli were given; *g* indicates galvanic curve; *r*, respiratory curve.

In studying the experimental results it was found that various types of galvanic reactions (based on their time relation to the stimuli) and the relative frequency of the types were characteristic features by which the records might be described, classified and evaluated. The responses which followed the stimuli closely (in from one to four seconds) were called direct reactions, figure 1 showing the galvanic and respiratory curves, the time record in seconds and the time of stimulation. If the responses occurred later than four seconds after the stimuli, they were classed as late reactions. Those appearing after an interval of eight sec-

onds were grouped as disconnected (fig. 2). Disconnected waves of lesser amplitude and occurring in groups were classed as spontaneous waves; they can be seen in figure 3 which presents part of a record taken when no stimuli were being given. Some of the stronger reactions were followed by after-fluctuations which are called secondary waves.

The foregoing differentiation into various types of waves considers only the individual reactions. In addition, it was found that the galvanic curves as a whole present a regular or a more irregular picture. I have therefore differentiated the records according to five degrees of regularity in shape, basing the classification on inconsistencies in the form

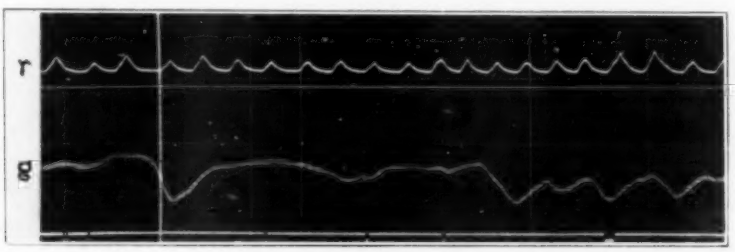

Fig. 4.—Galvanic reactions of irregular shape.

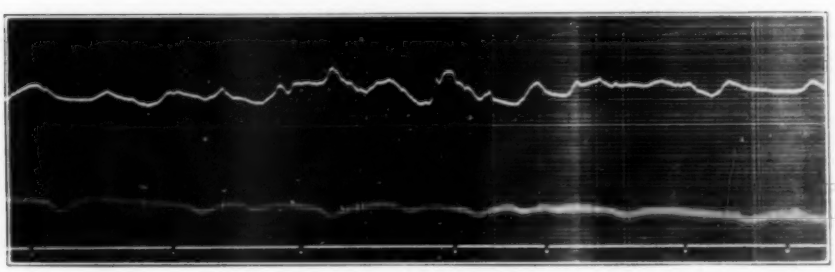

Fig. 5.—Irregular respiratory curve.

of the waves and disregarding variations in amplitude. The galvanic curve in figure 1 is regular in shape; that in figure 4 is irregular. The respiratory curves also were grouped into five degrees of regularity. Figure 5 shows a curve of irregular type.

The classification of the records made independently by me and by an assistant indicated that the divergencies of evaluation were relatively very small and that the personal factor, therefore, may be disregarded. A more detailed report concerning the technic of the experiment and the method of evaluation has been made elsewhere.[1]

1. Syz, H. C.: Psychogalvanic Studies on Sixty-Four Medical Students, Brit. J. Psychol. **17**:54 (July) 1926.

The purpose was to determine whether the records taken from persons belonging to the same clinical reaction type would show a certain correspondence and consistency in the features mentioned above. The observations from experiments performed on two fairly homogeneous groups of normal persons may be cited as a basis for comparison. The outstanding facts are given in figure 6 for both groups, the one con-

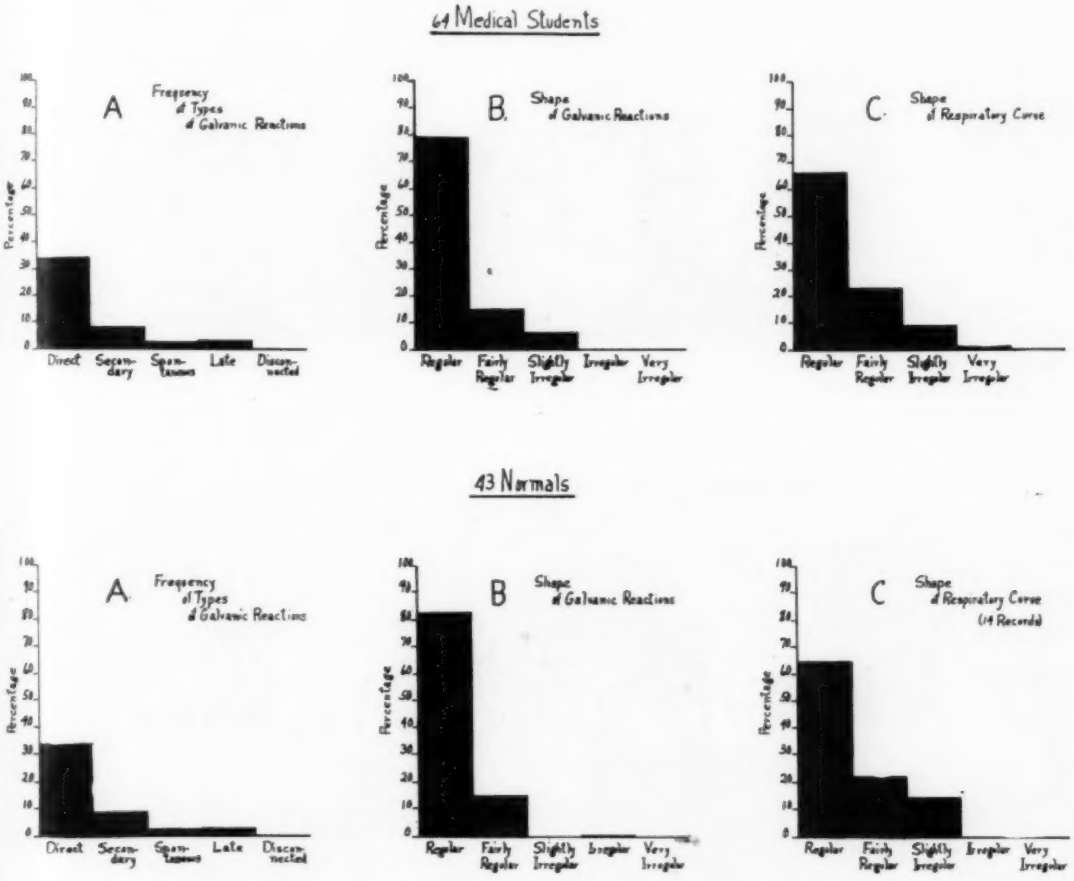

Fig. 6.—In two groups of normal persons: *A*, the frequency of the various types of galvanic reactions; *B*, the degrees of regularity of the galvanic, and *C*, of the respiratory curves.

sisting of sixty-four first year medical students, the other of forty-three persons including physicians, laboratory assistants and others not associated with the hospital. *A* shows the frequency of the various types of reactions, the height of the columns indicating (as per cent of the verbal stimuli given) the average frequency of responses throughout the entire group of persons. The various types of reactions are marked on the

horizontal axis. Direct reactions are predominant (34 per cent), late and spontaneous responses are not numerous (2 per cent and 3 per cent, respectively), and the disconnected fluctuations are almost entirely absent. The shape of the galvanic waves is tabulated in *B* and the shape of the respiratory movements in *C*. Both the galvanic and the respira-

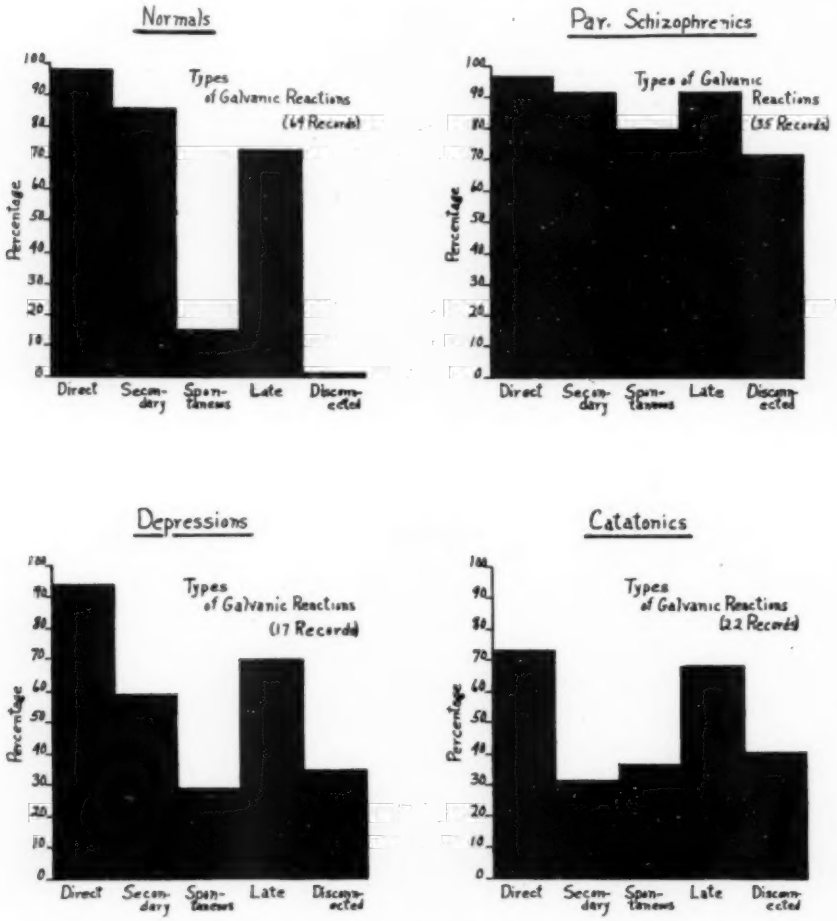

Fig. 7.—The percentage (on the vertical axis) of persons in each group who gave the various types of reactions (horizontal axis).

tory curves are predominantly regular, the tendency to slight irregularity being present in only a few cases. The fact that the results in these two groups of normal persons agree to such a large extent (as shown in the diagram) supports the contention that the averages thus obtained furnish a valid basis of comparison for individual cases as well as for other groups.

With these observations in normal persons I shall compare thirty-five records taken from twenty paranoid schizophrenic, twenty-two records from seven catatonic stupor and seventeen records from fifteen depressed patients (agitated cases were excluded [2]).

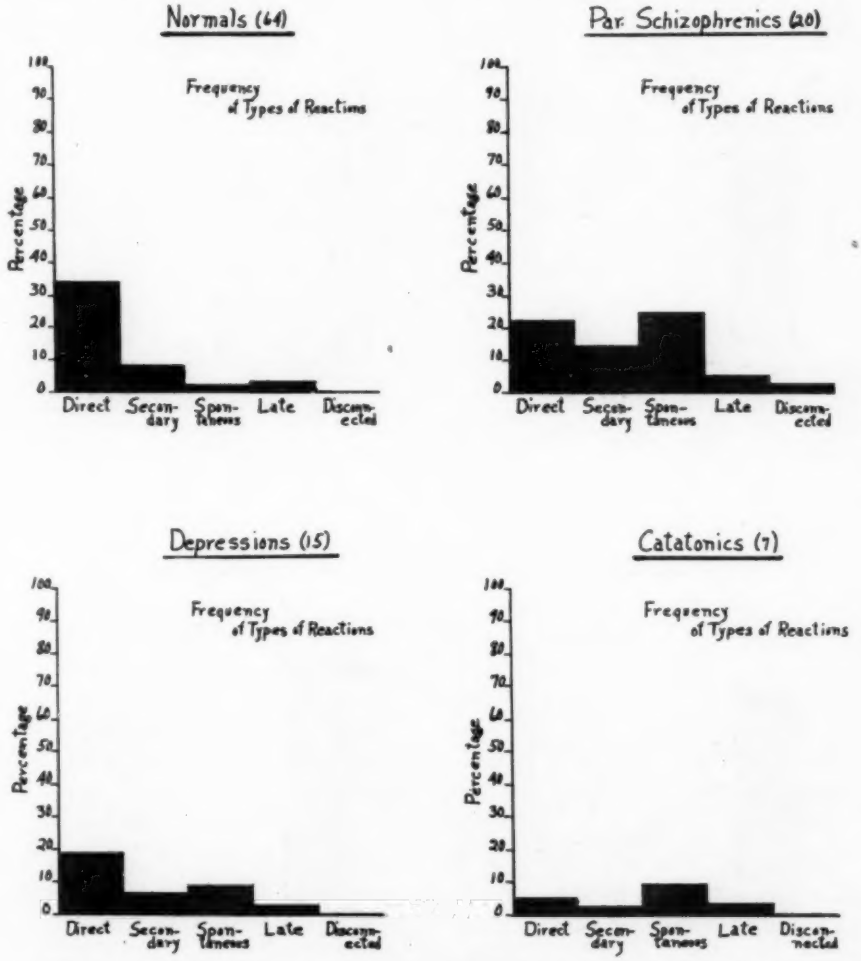

Fig. 8.—Average frequency of the various types of reactions in the four groups calculated as percentage of the verbal stimuli given.

Figures 7 to 10 give the comparative data for the four groups. Figure 7 indicates in how many persons of each group the various types of reactions were present. I wish to emphasize especially the fact that spontaneous and disconnected galvanic waves, present in only a

2. Differences in the galvanic curves in mental disorders have been described by other workers, Claparède, Gregor and Gorn, Peterson and Jung, Prideaux.

few normal persons, occurred in almost all paranoid schizophrenic patients (78 per cent), and also in a fair number of catatonic (38 per cent) and depressed patients (32 per cent). In figure 8 the average frequency of the various types of reactions is calculated for each group. This figure shows, even more clearly than the foregoing, the large num-

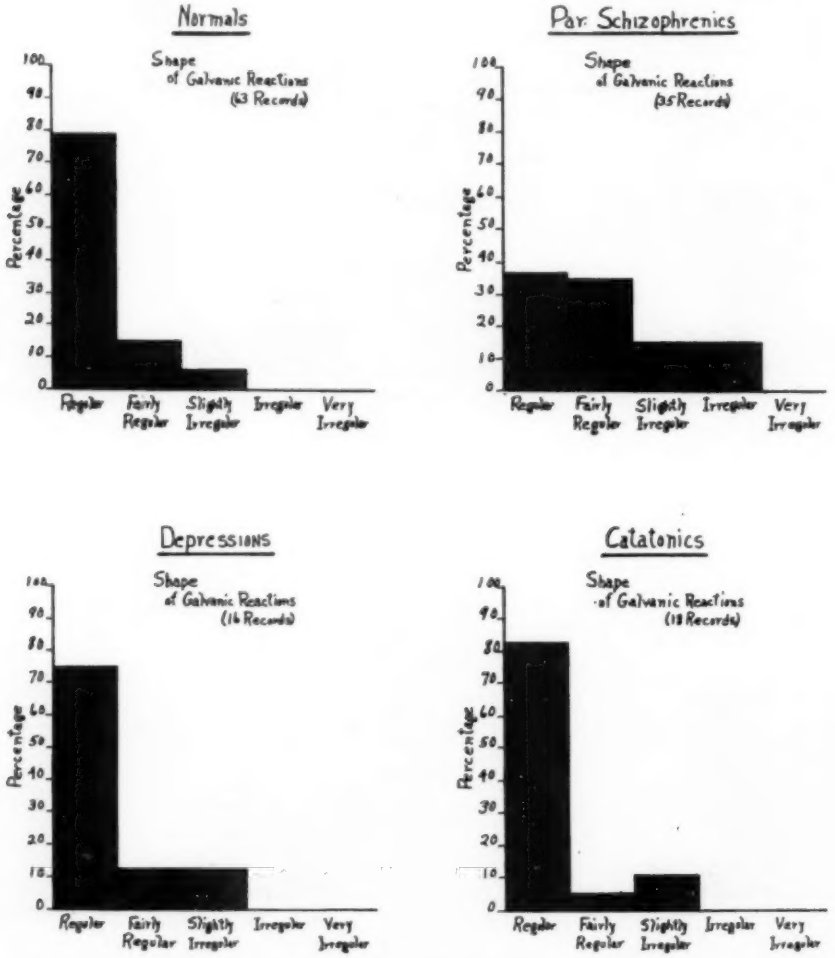

Fig. 9.—Classification of records according to the degree of regularity of the galvanic waves (as per cent of total number of records in each group).

ber of spontaneous and disconnected waves in paranoid schizophrenic patients, namely 23 per cent and 3 per cent, as compared with 2 per cent and 0.3 per cent in normal persons. The values for the catatonic and depressed fall between those for the paranoid and normal persons. The direct reactions occur in the schizophrenic patients less frequently (22 per cent) than in the normal persons (34 per cent), though the

former have on the average a higher total frequency of galvanic waves than the subjects of any other group here discussed. In the depressed patients the direct reactions are even less numerous (19 per cent), and in the catatonic patients, in whom the galvanic reactivity is generally much lower, they reach only 5 per cent.

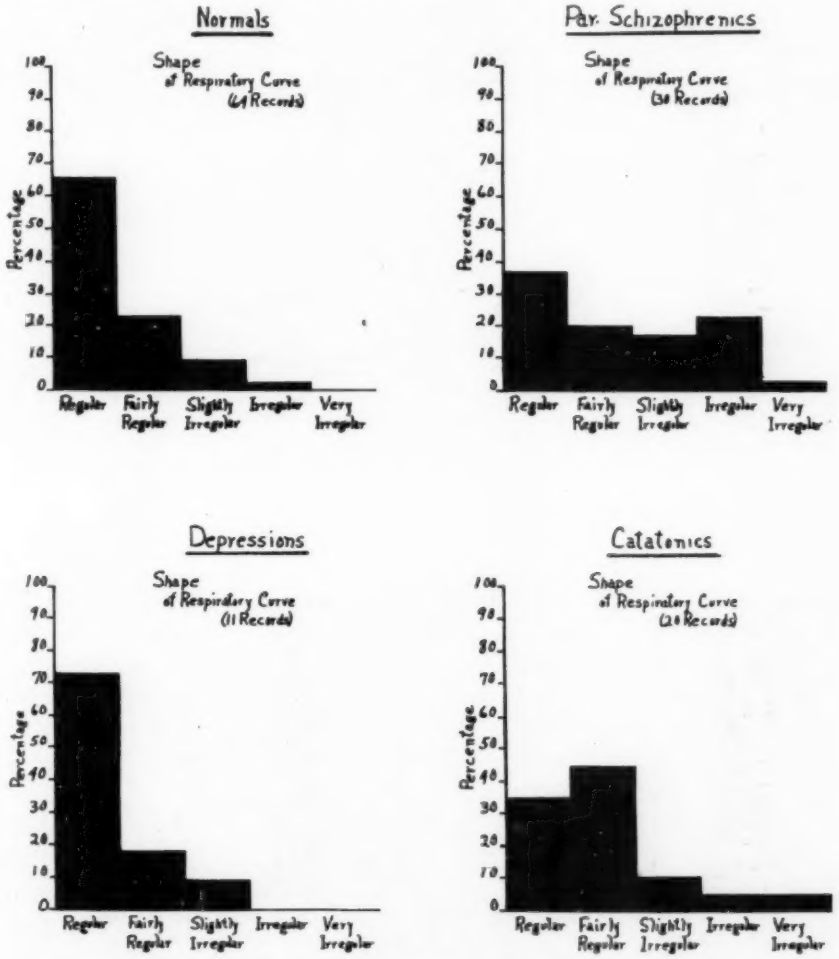

Fig. 10.—Classification of records according to the degree of regularity of the respiration (as per cent of total number of respiratory records in each group).

A comparison of the groups as to the shape of the galvanic curves (fig. 9) shows various degrees of irregularity in the paranoid schizophrenic patients as contrasted with normal persons and the depressed patients in whom only a few cases have the tendency to slight irregularity. We find a regular shape also in the catatonic patients in whom the reactions are on the whole much less numerous. The respiration

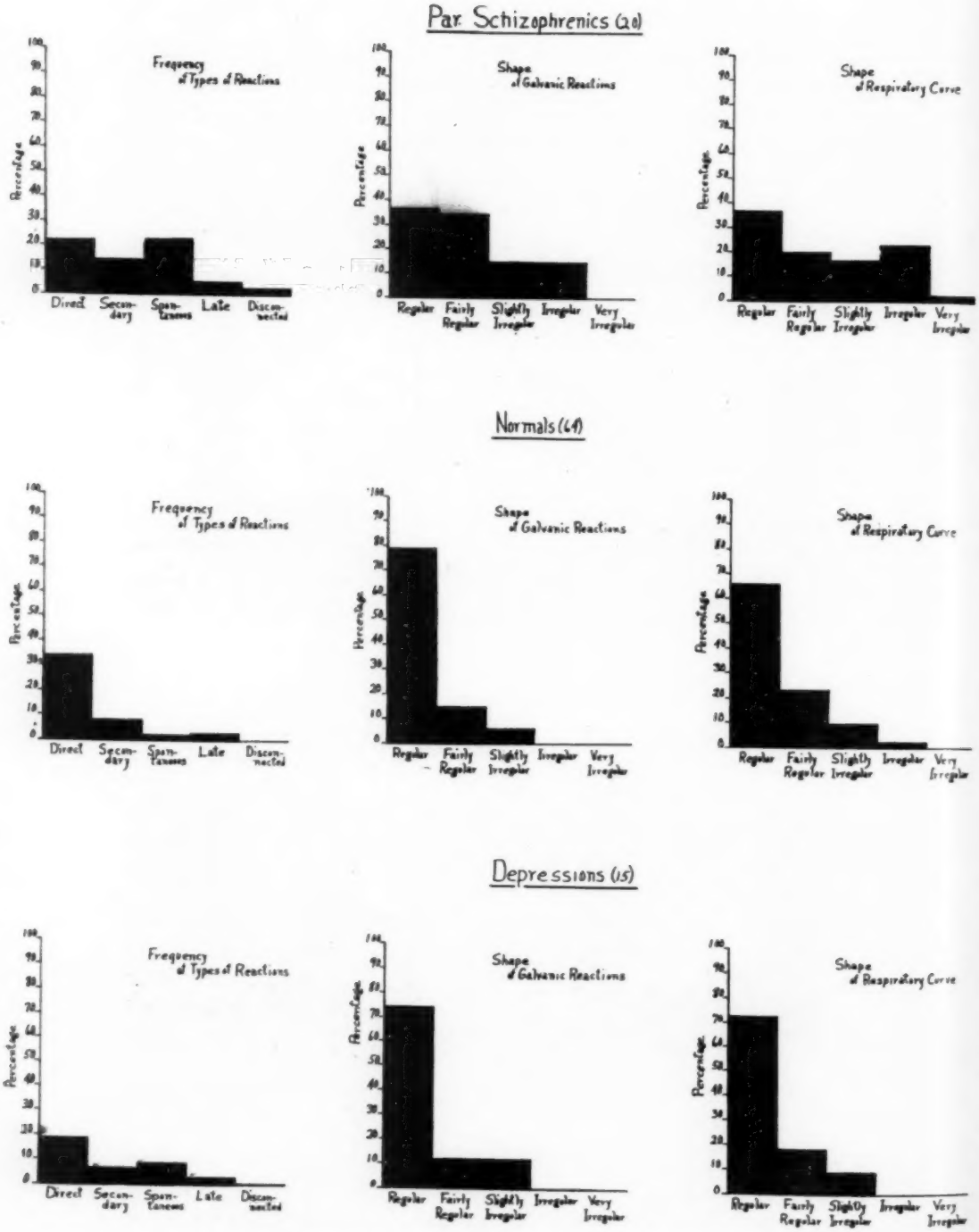

Fig. 11.—Comparison of the frequency of the types of reactions, the shape of the galvanic and of the respiratory curves in the groups of paranoid schizophrenic patients, normal persons and depressed patients.

(fig. 10) presents a slightly different picture, the catatonic as well as the paranoid schizophrenic patients tending to irregularity in this feature.

Figure 11 summarizes for comparison the aforementioned outstanding features as they appear in the three groups: paranoid schizophrenic

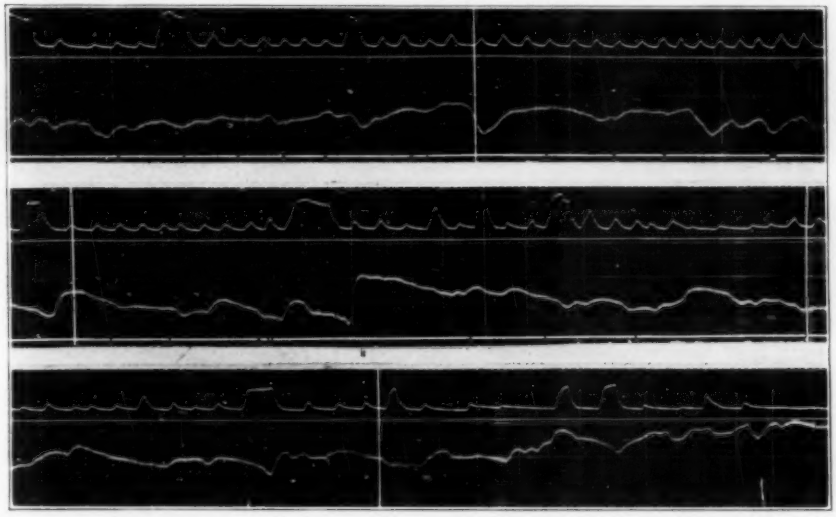

Fig. 12.—Record from a paranoid schizophrenic patient. Galvanic waves irregular and not closely connected with stimuli; respiratory curve irregular also.

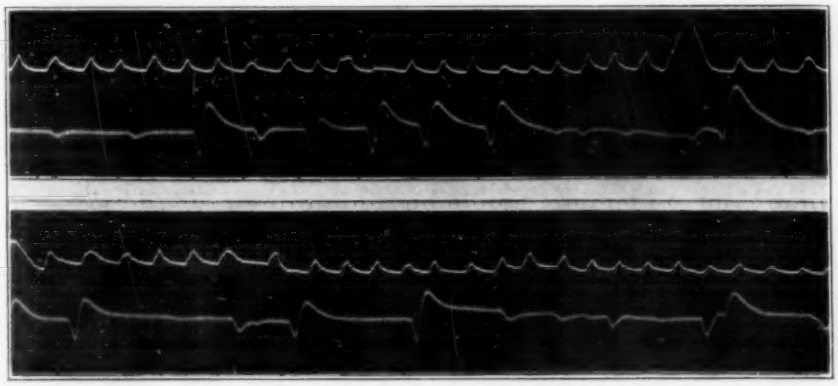

Fig. 13.—Record from a sensitive normal person. The galvanic reactions follow the stimuli directly and are of regular shape; the respiratory curve shows only slight irregularities.

patients, normal and depressed persons. In paranoid schizophrenic patients, in spite of their high total of galvanic waves, one finds fewer reactions which are closely connected with the outside stimuli but many waves which appear spontaneously, quite independent of environmental influences. This does not point to a lowered emotivity in these patients

but rather to the fact that their emotional fluctuations are directed by a type of stimuli other than that active in the average normal; it is an experimental corroboration of the clinical observation that these persons have the tendency to react (emotionally and in their behavior) more to endogenously arising stimuli and less to environmental situations. Besides, there is in schizophrenic patients a general tendency to irregularity of the galvanic as well as of the respiratory curve. One is tempted to see in these features a parallel to the incongruities of the emotional responses, to the unevenness of the affective life as it is clinically observed in these persons. Also in depressions the galvanic records correspond to what might be expected from a consideration of the clinical picture. In this group we have, with a low total number

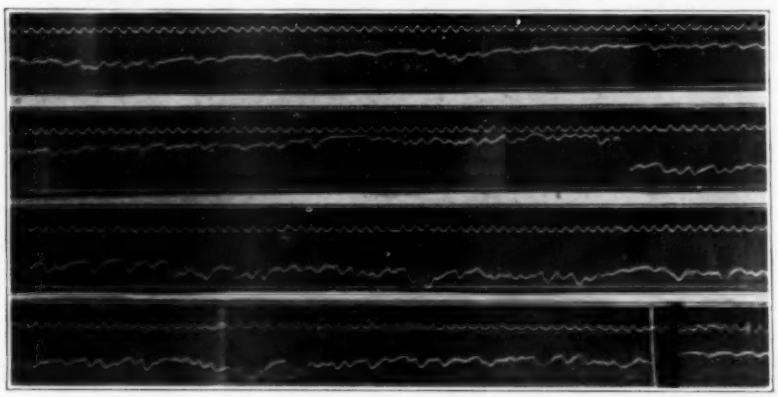

Fig. 14.—Record from a schizophrenic patient with pseudostuporous states. The galvanic waves are synchronous with the respiratory movements and show no connection with the stimuli.

of responses, an increase of the spontaneous waves and a consistent regularity in the shape of both the galvanic and the respiratory curves.

The differences between the records of normal and schizophrenic subjects presented in the preceding tables are further illustrated by figures 12 to 16 which show parts of individual records. Figure 12 is a section of a test taken from a patient who was markedly dissociated and detached, and who displayed great emotional unrest and incoordination along with a wealth of paranoid fancies. The galvanic fluctuations, which are numerous and of an irregular and inconsistent shape, show no direct connection with the stimuli (indicated on the base line). The respiration also presents marked irregularities. Such an outspoken and typical picture is not found in all paranoid schizophrenic patients, of course, but it illustrates what is the general tendency for patients of this group. Comparison with a record of a normal person (fig. 13) brings out the typical features even more clearly. In this very sensitive and

responsive person we find also frequent and strong responses, but they are closely connected with the preceding stimuli and are of regular shape (consistent in form). The respiratory movements show only slight irregularities.

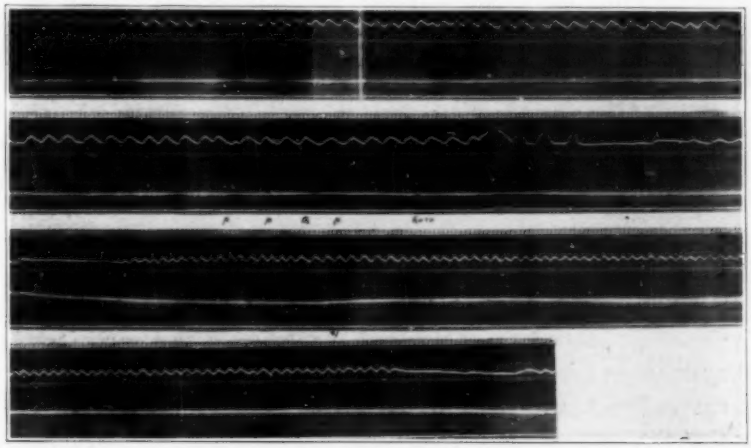

Fig. 15.—Record from a case of catatonic stupor. Galvanic curve flat; no responses to verbal stimuli, pin pricks (*p*), threat (*th*), horn or deep breathing; the patient took the deep breaths (following horn) at the suggestion of the experimenter; the short deflection in the curve with "horn" does not originate from the patient but is due to induction from the current used for sounding the horn.

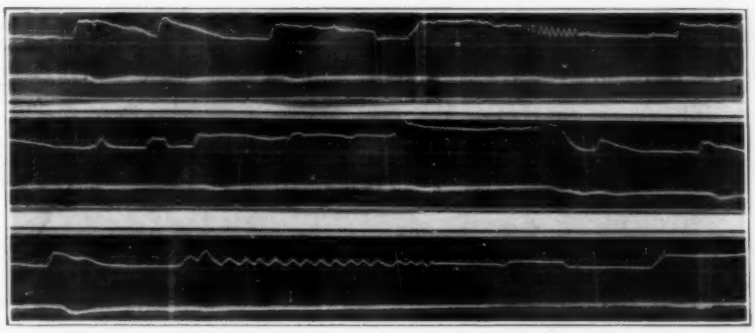

Fig. 16.—Record from the same patient as in figure 15. The galvanic curve shows only slight fluctuations; the respiratory curve is irregular and presents abrupt variations.

A more unusual curve obtained from a schizophrenic patient with semistuporous states is reproduced in figure 14. The numerous galvanic reactions are not definitely related to the stimuli but are synchronous with the respiratory movements, a feature not present in the characteristic curves of normal persons. This concomitance, which has also been

found in cases of catatonic stupor, shows a peculiar fusion of two otherwise relatively independent physiologic mechanisms.

The greatly diminished galvanic reactivity in catatonic stupors may be seen in figures 15 and 16. In this case (of catatonic stupor) even sensory stimuli (pin pricks, sounding of a motor horn) did not cause a deflection of the galvanometer string. This would indicate the patient's complete emotional detachment. Deep breathing also was not accompanied by galvanic waves (contrary to the observations in other patients and in normal persons). Whether this means a dissociation of two physiologic functions, which as a rule are in closer interrelation, or a blocking of the central (cortex) or peripheral (skin) mechanism, could not be determined. Another incongruity (fig. 16) is the occurrence simultaneously of abruptly changing respiration and low galvanic responsiveness as found in a number of catatonic patients.

Not all the persons included in this report gave records as typical as those presented in the figures above. One would expect variations in the psychogalvanic curves of persons who, though of the same reaction type, naturally vary in many behavior and emotional traits. We also know that the relationship between emotion and galvanic skin reflex may be influenced by a number of other factors which cannot be exactly estimated (local conditions of the skin, nervous influences not directly dependent on "mental" state, etc.). The galvanic records of persons of the same reaction type show, in spite of these complicating circumstances, features which are typical and fairly consistent. The general tendencies in the psychogalvanic (and respiratory) curves, as indicated in the diagrams in which the averages are calculated for each group, appear in each individual case, though in varying degree and combination. The psychogalvanic phenomenon, then, is of assistance in an objective, experimental discrimination and characterization of some of the outstanding psychopathologic reaction patterns.

THROMBOSIS OF THE SPINAL VESSELS IN SUDDEN SYPHILITIC PARAPLEGIA *

MON-FAH CHUNG, M.D.
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Interference with the blood supply of the spinal cord, excluding trauma, has long been recognized as the basis of sudden paraplegia. The lesion in syphilitic paraplegia is invariably thrombosis in the blood vessels, either intramedullary or extramedullary, producing thereby direct and secondary degenerative changes in the cord and giving rise to the apoplectiform symptoms. Although this view is now generally accepted, clear pathologic evidence for it is scarce. My purpose in this article, therefore, is not only to demonstrate the existence of actual thrombosed vessels and the distinctive type of softening which results directly from their occlusion, but also to show several concomitant changes in the meninges which, so far as I know, have not hitherto been described.

Bastian,[1] in 1882, was the first to express a definite opinion as to the cause of sudden paraplegia, known then as "acute myelitis," and maintained that "acute myelitis is in reality a degenerative softening resulting from vascular occlusion" in the region of the spinal cord, particularly in syphilitic lesions. About twenty years later, Singer [2] published pathologic observations, which strongly supported Bastian's contention. Spiller,[3] in 1909, recorded a case of thrombosis of the cervical median spinal artery, and demonstrated a degenerative process which involved the anterolateral aspect of the cord. Collins,[4] in 1912, described three cases of acute myelitis with evidences of softening from thrombotic vessels in one case, and agreed with Bastian that "acute myelitis is caused not by an inflammation but by thrombosis of some of the vessels of the spinal cord." Since then, Nonne,[5] Williamson,[6]

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* From the Department of Neurology, Peking Union Medical College.

1. Bastian, H. C.: Thrombotic Softening of the Spinal Cord as a Cause of So-Called "Acute Myelitis," Lancet **2**:1531, 1910.

2. Singer, H. D.: The Pathology of So-Called Acute Myelitis, Brain **25**:332, 1902.

3. Spiller, W. G.: Thrombosis of the Cervical Anterior Median Spinal Artery, J. Nerv. & Ment. Dis. **36**:601, 1909.

4. Collins, J.: Acute Myelitis and Thrombotic Softening of the Spinal Cord, Rev. Neurol. & Psychiat. **10**:157, 1912.

5. Nonne, Max: Syphilis und Nervensystem, ed. 3, Berlin, S. Karger, 1921.

6. Williamson, R. T.: Diseases of the Spinal Cord, Oxford Med. Pub., 1908.

Schultze,[7] Cadwalader,[8] Buzzard and Greenfield,[9] and others have voiced the same opinion, and it may be of interest to quote Spiller's recent emphatic statement [10] that "a rapidly developing paralysis of the lower limbs, or of all four limbs, is suggestive of thrombosis of the vessels of the spinal cord, and the resulting lesion is one of myelomalacia. It is not an infrequent development of spinal syphilis, and when it occurs in such cases it is still the result of thrombosis of the vessels of the cord sclerotic from syphilis; the resulting myelomalacia is associated with the inflammatory lesions of syphilis."

I have recently called attention to the frequency of sudden paraplegia in syphilitic Chinese patients,[11] and now wish to present two cases in which the vascular lesions are clearly demonstrated—the one involving especially the arteries, and the other only the veins, producing identical changes in the cord in each case.

REPORT OF CASES

CASE 1.—An unmarried rickshaw coolie, aged 35, was brought to the Peking Union Medical College Hospital, May 10, 1921, with complete paralysis of both lower limbs and retention of urine, which had developed suddenly in the night a week before admission. On the day before the onset, he pulled his rickshaw in the rain. He gave a history of gonorrheal infection seven years previously with a rather obscure primary lesion.

Examination.—This showed loss of all forms of sensation from a level corresponding to the seventh thoracic segment to both soles, a flaccid type of paralysis, absence of superficial and deep reflexes below this level, and no Babinski reflex or clonus. Between the fourth and seventh thoracic levels there was a zone of hyperesthesia. The sphincters were spastic. No bedsores or trophic changes were found. Both blood and spinal fluid Wassermann reactions were strongly positive.

Course.—While in the hospital the patient first showed a few signs of improvement, but soon developed bedsores and evidences of cystitis, from which he died four months after entry.

Necropsy.—This was refused by the authorities, but the cord was fixed in situ with solution of formaldehyde. A month later, the cord was removed in the anatomic laboratory. Macroscopically, except for a slightly milky appearance of the cord, nothing of special importance was recorded. Between the first and third thoracic segments the cord appeared soft and shrunken.

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7. Schultze, F.: Ueber Myelitis akuta und akutissima . . . , Deutsche Ztschr. f. Nervenh. **68**:1, 1921.

8. Cadwalader, W. B.: Onset of Spinal Paralysis, Arch. Neurol. & Psychiat. **6**:541 (Nov.) 1921.

9. Buzzard and Greenfield: Pathology of the Nervous System, New York, Paul B. Hoeber, 1922.

10. Spiller, W. G.: Sudden Paraplegia, Arch. Neurol. & Psychiat. **13**:471 (April) 1925.

11. Chung, M. F.: A Study of Thirty-Four Cases of Rapidly Developing Syphilitic Paraplegia, Arch. Dermat. & Syph. **14**:111 (Aug.) 1926.

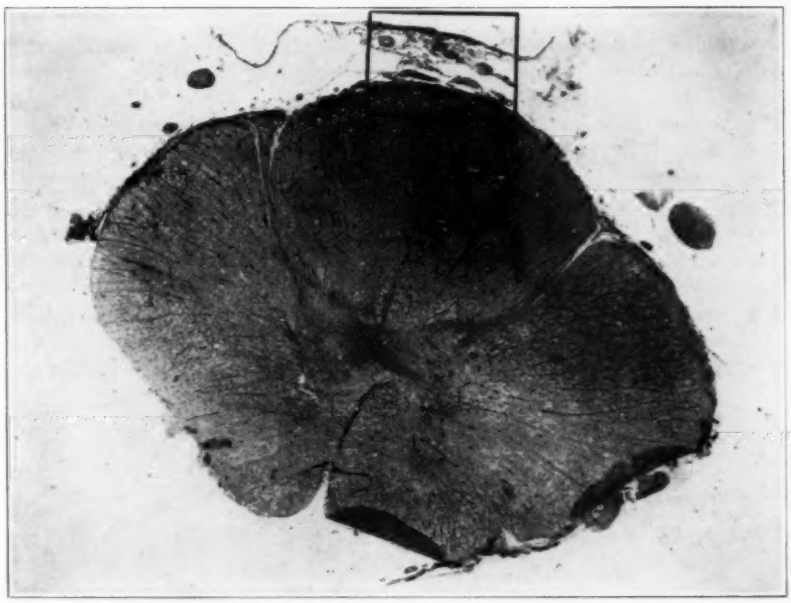

Fig. 1 (case 1).—Section of cord at level of second thoracic segment. Demarcation indicates position of thrombosed vessels. Extensive degeneration throughout section shown clearly in Weigert's stain. Van Gieson stain; × 7.

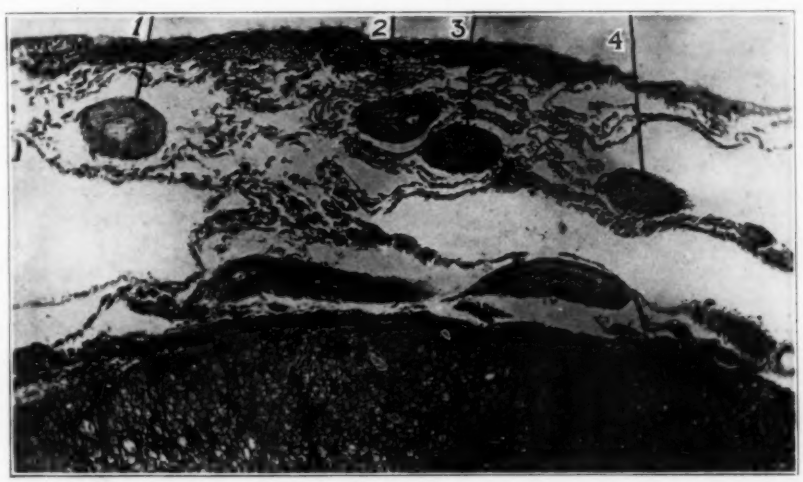

Fig. 2 (case 1).—Section from the posterior aspect of the cord showing four arteries in various stages of thrombosis: *1*, partial obstruction with much thickened vessel wall; *2*, canalization; *3* and *4*, complete occlusion. Exudate in meninges chiefly fibrin, endothelial cells and a few lymphocytes. Van Gieson stain; × 70.

Microscopic Examination.—At the site of the lesion (second thoracic segment), there was extensive destruction of the cord substance with slight meningeal changes. The degeneration of the white matter was most marked peripherally, in places the neural elements being completely disintegrated and replaced by masses of large cells with small, deeply stained, round, eccentric nuclei and pale honeycombed cytoplasm, not unlike the "gitter" cells described by Jacob and associated with myelin degeneration.[12] Where the softening was less marked, only a network of glia tissue remained with many vacuoles (both myelin and axons being absent), often filled with one or more gitter cells. The vascular elements seemed increased, but except for the infiltration of round cells in the vessel walls, there was no evidence of hemorrhage or thrombosis. Moderate glia proliferation was seen throughout the section. The ganglion cells in the gray matter all showed varying degrees of chromatolysis, from acute swelling to a shriveled mass.

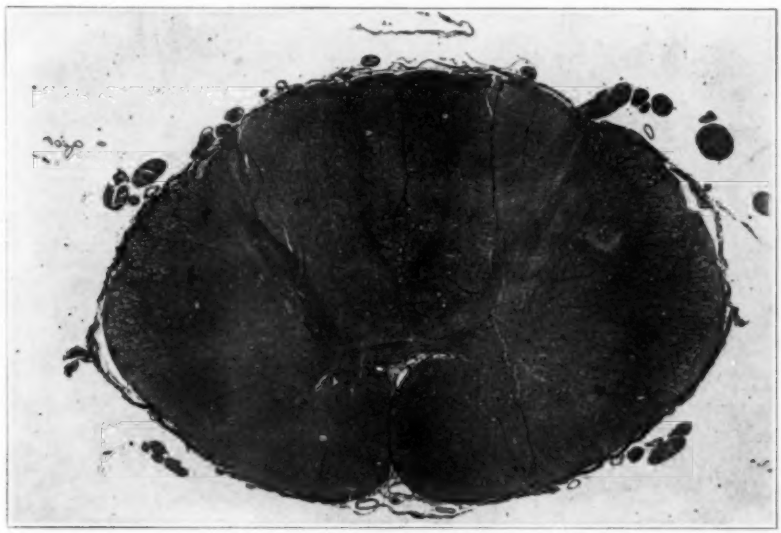

Fig. 3 (case 1).—Section of the cord at cervical level, showing ascending degeneration of tracts, with relatively normal appearance of the meninges and the blood vessels on both the anterior and posterior aspects. No evidence of exudate but only a slight round cell infiltration in the meninges. Van Gieson stain; × 7.

The meningeal thickening was slight except on the dorsal aspect where there was moderate proliferation of the connective tissue elements, together with some infiltration of round, plasma and endothelial cells. It was on this part of the cord that thrombosis of the arteries (fig. 1) in various stages of organization, from complete block to canalization, was found. The group of vessels, particularly the arteries, just dorsal to the posterior septum was chiefly, and almost solely, affected; elsewhere about the cord the vessels appeared normal; in addition, the intimal coats were much hypertrophied and infiltrated with round cells. The inflammatory exudate was slight.

12. Jacob, A.: Ueber die feinere Histologie der sekundären Faserdegeneration in der weissen Substanz des Rückenmarks, Nissl-Alzheimer's Arb. **5**:1, 1912, quoted by Bassoe and Hassin, footnote 13.

Above the lesion, with the exception of the secondary ascending degeneration of the tracts and some chromatolysis of the anterior and posterior horn cells, the cord, meninges and blood vessels showed little evidence of pathologic change.

Below the lesion, down to the sacral portions, the fiber tracts showed descending degeneration, but a noteworthy change was found in the pia-arachnoid as compared with the condition above the level of the lesion. The pia-arachnoid on the posterior and dorsolateral aspects of the cord was thickly infiltrated with cells with considerable proliferation of the connective tissue elements. An inflammatory exudate, consisting of many polymorphonuclear, large mononuclear, round, plasma and endothelial cells, with fibrin, was found. The meningeal vessels were dilated, and some were congested with blood cells, with marked thickening of

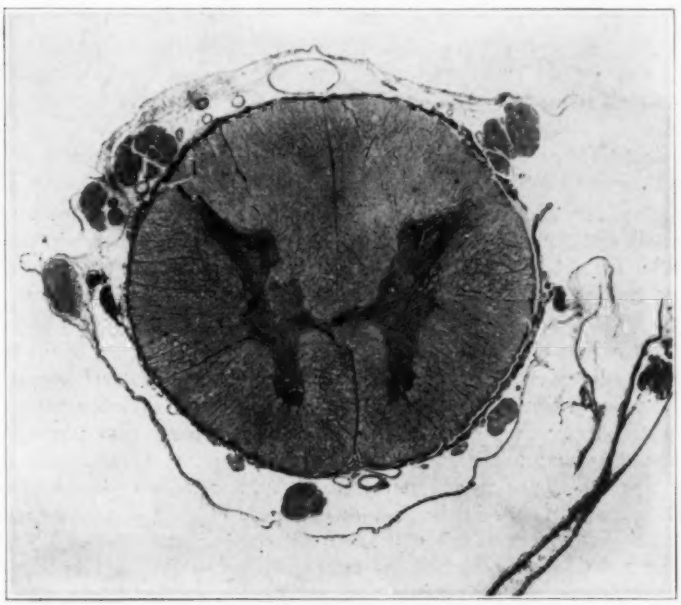

Fig. 4 (case 1).—Section of the cord at lower thoracic level, showing descending degeneration of tracts, and distended meninges, marked cellular infiltration, chiefly polymorphonuclear leukocytes, lymphocytes and endothelial cells, and the dilated blood vessels especially on the dorsal and dorsolateral aspects. Van Gieson stain; × 6.

the intima of the arteries and slight endovascular and perivascular infiltration of round cells. The blood vessels on the ventrolateral and anterior aspects were only slightly dilated or infiltrated with cells.

This was a case of sudden paraplegia in a young man, who on admission showed complete flaccid paralysis of both lower limbs with sensory disturbances, spastic sphincters and laboratory evidences of syphilis. Trophic changes and other complications set in, which brought about death. The pathologic changes included thrombosis of the posterior spinal meningeal arteries with direct softenings and secondary degen-

erative changes in the cord, and evidences of an acute inflammatory exudate in the meninges at and below the lesion, especially on the posterior and dorsolateral aspects of the cord.

CASE 2.—An unmarried merchant, aged 26, was admitted to the hospital, Feb. 29, 1924, complaining of inability to walk and to pass urine. He denied syphilitic infection. The onset of the present illness had been sudden. On awakening one morning, he was unable to move his legs; he had had some difficulty the previous night with urination, which progressed to complete retention.

Examination.—This revealed a zone of hyperesthesia corresponding to the eighth, ninth and tenth thoracic levels, but from the eleventh thoracic down to the sacral level the anesthesia was complete for all forms of sensation. Both lower extremities were paralyzed with all reflexes lost. There was no Babinski sign or clonus, and the sphincters were spastic. Bedsores were present on both buttocks. Blood and spinal fluid Wassermann reactions were strongly positive, and in addition the spinal fluid gave a cell count of 40, a protein content of 1.16 mg. per cubic centimeter and a colloidal curve of 3333221000.

Course.—In spite of vigorous antisyphilitic treatment, the cystitis developed into a pyelonephritis which caused death nine weeks after entry.

Necropsy.—The brain and cord were removed. The gross appearance of both was not striking; the meninges appeared less transparent than normal and were covered with a light brownish discoloration in the medulla and cervical portions. The upper end of the thoracic region was soft to the touch. There was no congestion of the vessels.

Microscopic Examination.—Histopathologic study of the cord about the third thoracic segment revealed extensive disintegration of the neural elements with large areas of softening, consisting chiefly of gitter cells, involving the peripheral as well as the center of the cord. These gitter cells were also found scattered in the white substance in which the degeneration was less severe. The areas of softening appeared wedge-shaped at the periphery, and more or less oval near the center. The ganglion cells in the gray matter were reduced in number, and those remaining showed chromatolysis and slight neuronophagia. The vascular supply appeared increased; no hemorrhage or thrombosis was found, but in many of the vessel walls could be seen a moderate amount of infiltration with round cells. The glial elements were somewhat increased, particularly about the softened areas.

The meninges about the lesion were only slightly thickened and infiltrated with lymphocytes and a few plasma and endothelial cells, but no polymorphonuclear cells. The vessels in the pia, especially the veins on the dorsal and lateral aspects, showed thrombosis and round cell infiltration in the intima of the vessel walls (fig. 6). The occlusion of the vessels by organized fibrin, cells and pigmented particles could be seen to affect chiefly the venous supply. The arterial walls did not reveal any hypertrophy or much cellular infiltration.

Above the lesion, in the cervical region, there was ascending degeneration in the fiber tracts, and chromatolysis of the ganglion cells in the gray matter. The meninges were not thickened, and the cellular infiltration was moderate. The blood vessels in the cord as well as in the pia did not appear dilated or congested; but here again, the veins in the latter showed only small round cell infiltration in the thickened muscular walls, especially the dorsal group.

Below the lesion, at the level of either the lumbar or sacral segments, besides the descending degeneration of the tracts and ganglion cells in the gray matter, there was evidence of an inflammatory exudate in the meninges, especially in the

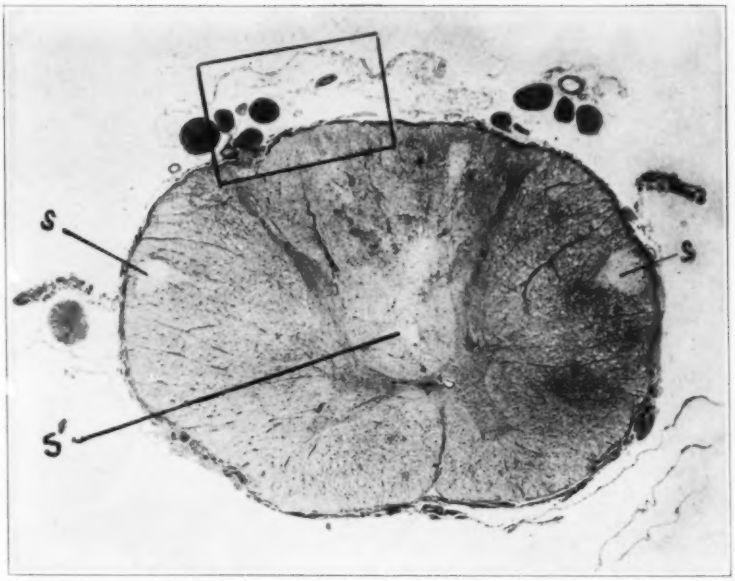

Fig. 5 (case 2).—Section of cord at level of third thoracic segment. Insert enlarged in figure 6 shows a few of the thrombosed vessels: *S*, wedge-shaped softening at periphery; *S'*, softened areas near the center. Van Gieson stain; × 7.

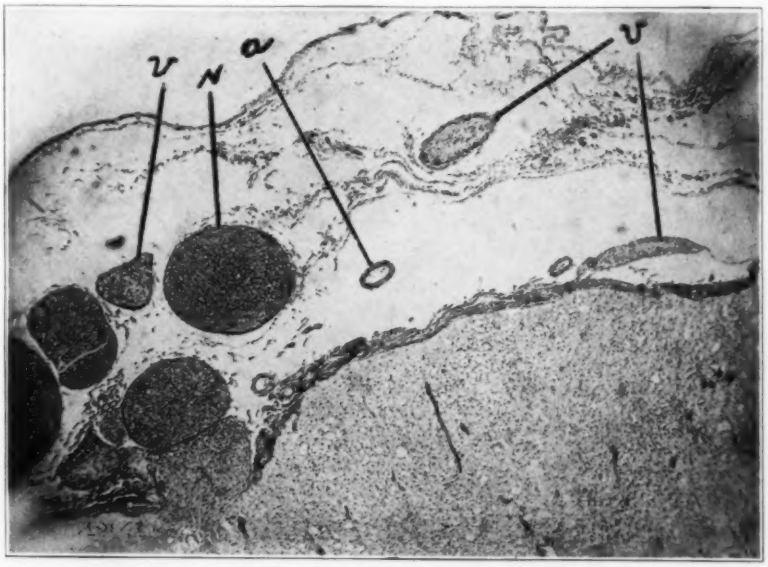

Fig. 6 (case 2).—Enlargement of figure 5: *v*, thrombosed veins; *a*, normal artery; *N*, posterior nerve root. The slight exudate in the meninges, consisting largely of fibrin and endothelial cells and a few small round cells, should be noted. Van Gieson stain; × 60.

Fig. 7 (case 2).—Another view of the thrombosed veins, showing the artery lying beside the lower one and the round cell infiltration in the wall of the thrombosed vessels. Van Gieson stain; × 70.

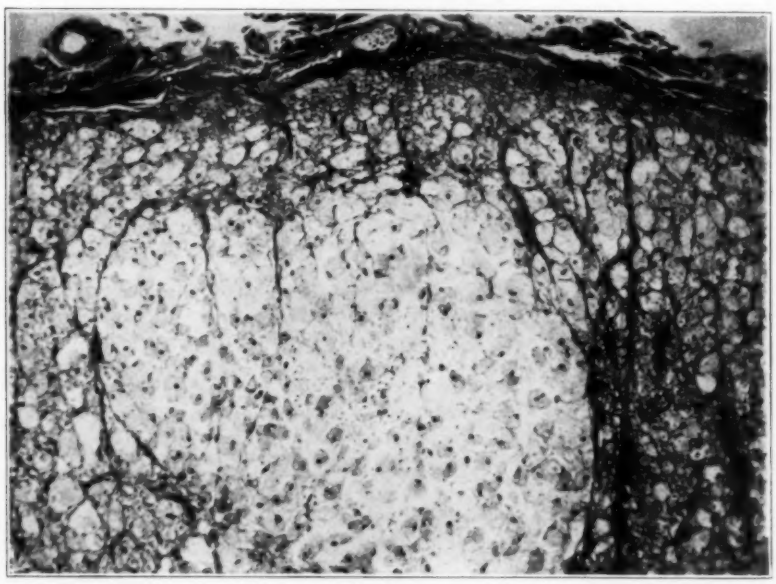

Fig. 8 (case 2).—A patch of softening, showing the mass of gitter cells and the absence of round cells in it. On the right margin of the softened area, there is a moderate glia proliferation. Van Gieson stain; × 70.

dorsal aspect, consisting chiefly of small lymphocytes and a few plasma and endothelial cells, and large mononuclear leukocytes, fibrin and dilated congested blood vessels. The pia-arachnoid and the veins were considerably hypertrophied and thickly infiltrated with lymphocytes, the arteries being less affected.

This young man, with a history of an acute onset of paraplegia and retention of urine, was found to have a complete transverse lesion of the cord with sensory and reflex disturbances from a level corresponding to the eighth thoracic to the sacral segments. Both blood and spinal fluid examinations pointed to syphilis. Death from intercurrent infection followed after a few weeks in the hospital. Necropsy revealed periphlebitis and endophlebitis with thrombosis of the pial vessels on the dorsal and lateral aspects of the third thoracic segment, resulting in softening of the nerve tissue at that level and secondary changes above and below the lesion. In addition, there was also meningeal reaction of an inflammatory character below the lesion, but no such changes in the membranes above. The exudate, as far as the meninges were concerned, was confined more to the posterior aspect of the cord than to the anterior region.

COMMENT

The pathologic evidence presented leaves little doubt as to the factor of thrombosis in the production of sudden paraplegia. With the stoppage of the blood supply of one or more important vessels in the pia of the spinal cord, as in the brain or elsewhere in the body, there will result paralysis of function corresponding to the location of the infarction. Foci of such softening, filled with gitter cells and practically no lymphocytes other than a few about the vessels, are found in sections in which the thrombotic vessels appear (fig. 5). The infiltration with small round cells, although seen within the intima of many of the small vessels in the cord, has not extended to the surrounding tissues in sufficient amount to be regarded as a true myelitis. One may look on the degenerative process in the parenchymatous tissue as noninflammatory, a myelomalacia not unlike that described by Hassin [13] in his case of typical "yellow" softening of the cord. Singer,[2] in his illustrations, clearly showed such patches of softening about small thrombosed vessels with absence of leukocytic infiltration in the contiguous tissue; while in Collin's photomicrographs, similar areas of softening were demonstrated associated with diseased blood vessels and in one case with thrombosis of some of the smaller arteries. Neither of these writers made any mention of the condition of the meningeal vessels. Judging from the wedge-shaped foci of softening, scattered at the peripheral parts of the cord, shown in Collin's as well as in the accompanying illustrations, I

13. Bassoe, P., and Hassin, G. B.: Myelitis and Myelomalacia, Arch. Neurol. & Psychiat. **6**:32 (July) 1921.

believe that the lesions in his cases could have been accounted for by thrombosis of the radiating vessels entering from the pia-arachnoid.

These wedge-shaped foci of softening consisting of gitter cells and neural detritus, when found to be noninflammatory, might be regarded as significant of some circulatory disturbances, either from emboli or thromboses of the coronal vessels of the cord. Pick,[14] in his pathologic studies of caisson disease, demonstrated such wedge-shaped softenings at the periphery of the cord, and attributed them to anemic and ischemic processes. No explanation was advanced by him as to why peripheral softenings assumed such forms. Obersteiner[15] explains them on the basis of the branching of the coronal vessels in the substance of the cord. He believes that the radiating arteries, as they penetrate the white substance, spread out like a pine-tree (after Mager) with the top pointing centrally. If this were the only factor, we should find a similar form of softening following the occlusion of vessels at a point nearer the center, say at the middle third portions. However, as foci of softening centrally tend to an ellipsoid or spherical form, I believe that the internal structure of and the physical factors within the cord more or less determine the shape of the softening process, as pressure from the intact septums would force a semigelatinous mass to assume an oval shape centrally and a wedge peripherally. If such sector-like foci at the periphery may result from occlusion of the extramedullary vessels, then central ellipsoid softenings are produced by intramedullary lesions.

The meninges, however, present a singular picture. Their fairly normal appearance, excepting the slight infiltration of small round cells, especially in the blood vessels, above the lesion stands out in marked contrast to the portions at and particularly below the lesion. The exudate of cells, largely polymorphonuclear, lymphocytic and endothelial in types, the increase of fibrin and the congested, dilated blood vessels indicate some inflammatory reaction. But why should it be confined to the lower portion of the cord? I think the answer may be found in that, just as there is a generalized trophic change elsewhere in the body below the lesion, so also the meninges of the cord caudad to the thrombotic vessels share in the general vasomotor and trophic disturbances and decreased resistance.

The pathologic observations, shown here in regard to the distribution of the thrombotic vessels and the inflammatory exudate, appear to support Nonne's statement[5] that "specific meningitis is by far most frequently located on the posterior surface of the cord." The dorsolateral group of vessels, being smaller in caliber than the anterior median group, are perhaps more vulnerable and readily occluded in

14. Pick, A., in Flatau's Handbuch der pathol. Anatomie des Nerv. Syst. **2**:863, 1904.

15. Obersteiner, H.: Nervöse Zentralorgane, 1911, pp. 348-349.

syphilitic arteritis or phlebitis. When the specific virus attacks the vessels chiefly and hardly affects the meninges, the inflammatory reaction would most likely gather in greater amount about the diseased vessels. Such an explanation would seem plausible in the cases described above. As similar increased exudate of the posterior aspect of the cord (if the extent of the meningitis is determined by the amount of the inflammatory exudate) is observed in tuberculous or meningococcic meningitis, it does not appear to be distinctive of syphilitic meningitis. I feel, however, that in these other types of meningitis, as also in chronic syphilitic meningomyelitis in which the exudate is dense and rich, the influence of gravity in the distribution of the exudate must be borne in mind, for as a rule patients suffering from these chronic diseases are bedridden for weeks or months.

Nonne is of the opinion that thrombosis of both the larger and smaller spinal arteries and also thrombosis of the veins is of common occurrence. My experience somewhat supports his statement. However, in scanning the literature, I find only one report wherein the veins alone suffered. This record was made by Lamy,[16] in 1893, when he described a case of acute myelitis showing pathologic changes in the veins. One of the two cases reported here is probably the first on record to show both phlebitis and actual anatomic proof in the form of sections of the thrombosed vessels themselves.

CONCLUSIONS

1. Thrombosis of one or more of the important spinal vessels is the mechanism of sudden syphilitic spinal paraplegia.

2. Either the arteries or the veins alone, though usually both together, may become affected, while little or no meningitis or myelitis occurs.

3. Foci of softening, filled with gitter cells and noninflammatory in character, follow such thromboses—those of wedge shape at the periphery from stoppage of the radial vessels, and those of ellipsoid forms at the center from occlusion of intramedullary ones.

4. In addition to the changes in the cord, there is also a meningeal reaction below the lesion in the form of a mild inflammatory exudate, which is explained on the basis of vasomotor or trophic disturbances.

5. The frequency of the involvement of the posterior aspect of the cord as compared with the anterior may be accounted for by the relative size of the blood vessels and their vulnerability to the attack of the syphilitic virus.

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16. Lamy, quoted by Nonne, footnote 5.

Abstracts from Current Literature

MALARIAL TREATMENT OF MULTIPLE SCLEROSIS. G. L. DREYFUS and R. HANAU, Deutsche med. Wchnschr. **52**:354 and 391 (Feb. and March) 1926.

Since Wagner-Jauregg introduced malaria inoculation in general paralysis, in 1917, this treatment has found its way to all therapeutic centers, and the excellent results obtained by the method in general paralysis have prompted its use in other diseases of the nervous system, especially in multiple sclerosis. As shown by Steiner and Kuhn, multiple sclerosis appears to be an exogenous infectious disease, with an individual causation. The first experiments with fever in cases of multiple sclerosis were made in the clinic of Wagner-Jauregg. Gross treated multiple sclerosis with staphylococcus vaccine, later with typhoid vaccine. In all, Gross treated fifty-nine patients with vaccine (twenty with typhoid vaccine). The number of fever attacks provoked, as a rule, was from six to ten. It was endeavored to bring the fever to more than 38 C. at least six times. In some cases vaccine was combined with neo-arsphenamine. Gross noted vast improvement in 6 per cent of his chronic cases, and since the best results were obtained in cases in which typhoid vaccine was combined with neo-arsphenamine, he believes this combination to be preferable. The shorter the duration of the disease process, the greater was the improvement.

Gross inoculated forty-two patients with malaria virus. Most of them received neo-arsphenamine in addition to the malaria. The number of malaria attacks was from eight to ten. In some cases results immediately followed the treatment, and in others vast improvement was noted some time afterward. Brilliant results were obtained in 26 per cent of all patients treated, including those with acute forms of the disease. In 43 per cent the improvement was somewhat less. In only 16 per cent of chronic progressive cases was there marked improvement, while 41 per cent showed only slight improvement. According to Gross, the malaria therapy is not quite so good as the typhoid treatment, although one of his patients reacted better to malaria than to typhoid virus.

A follow-up of the treated patients showed an inclination to further improvement in those who were dismissed markedly improved. In other cases the disease slowly progressed after a shorter or longer period of continued improvement.

Typhoid Treatment.—For the vaccine, a very old laboratory strain was used. Twenty-four hour agar cultures were deposited in physiologic sodium chloride solution and the number of typhoid bacteria was determined according to the method of Wright. The solution was heated for an hour and a half to 56 C. The sterile solution was then thinned out with saline solution so that there were 350 million germs in each cubic centimeter. This solution was placed in sterile ampules of 5 cc. and left for twenty-four hours. Then each ampule was again sterilized. The saline solution used to thin out the deposit contained no disinfectant.

The initial dose was 0.01 cc., according to Schacherl, combined with 5 or 10 cc. of a 10 per cent calcium chloride solution. After three, four, five or

six injections, 0.3 or 0.45 Gm. of neo-arsphenamine was combined with the mixture, depending on the case. Some patients reacted with a high temperature, while others did not react to so slight a dose, and in these cases the dosage was immediately greatly increased.

The desired fever was manifested by chills, rigidity of the entire body, exhaustion, headache and often vomiting. The fever occurred from one-half to one hour after injection. The temperature then gradually mounted to 38 or 39, sometimes 40 C., within three or four hours, according to the dose and the individual case. In general it decreased after from five to eight hours, so that within ten or twelve hours it was normal. As a rule, however, the temperature did not return to normal the same day, but even the day following injection there was slight increase and only on the third or fourth day was the temperature normal. The patient's discomfort during the fever was not great. Headache and drawing pains throughout the body were the chief complaints. Pulse acceleration was noted according to the increase of temperature. After a period of normal temperature for three or four days the vaccine injections were again given. In the course of the treatment from ten to twenty injections were given, so that about twenty injections were given during one period of treatment of from eight to ten weeks. The general condition of the patient, including weight, was scarcely disturbed during the treatment period.

Of the authors' seven chronic progressive cases, two showed improvement. In one patient with a rapidly progressive polysclerosis the disease was arrested, and in the following seven months he did not grow worse. Four remained unchanged and did not grow worse during the period of observation. One of these patients had been observed by the authors for half a year before the therapy was used. He had been treated with albumin and arsenic without benefit. He improved so greatly by the typhoid vaccine therapy that for the past six months he has been able to resume his work as carpenter. All the cases reported were observed for from three to nine months after the treatment was discontinued — a very short time for this chronic disease, yet long enough to show that if improvement did not take place immediately after treatment it could not be brought about later.

In summarizing the results of the treatment the authors believe them to be better than those obtained by other therapy, and while not entirely satisfactory, certainly enough to encourage further experiments with fever treatment.

Malaria Treatment.—The malaria strain employed, which is biologically a tertian parasite, has been kept up through more than sixty inoculations. This strain has proved satisfactory, as it is sensitive to quinine. Injections were usually given intravenously, from 2 to 4 cc. of malaria blood being slowly injected. Anaphylactic reactions were not noted by this method. The period of incubation seems to be the same whether 2 or 5 cc. are inoculated. Naturally the incubation period is longer with intramuscular inoculations. In the authors' cases it was fourteen days. With intravenous inoculations it varied from four to nine days.

The elevation of temperature usually occurred in from one to three hours and was frequently accompanied by severe chills, sometimes of one and one-half hours' duration. The course of the malaria varied in each patient. In some cases the temperature returned to normal in a few hours and increased again in from twelve to eighteen hours. It also happened that the temperature remained between 39 and 40 C. for from three to five days. Such cases required

careful watching. One of the authors' patients had nine, two had ten, two had twelve, three had thirteen and one had fifteen and another twenty attacks of fever.

The effect of the fever on the general condition of the patient depended on the duration and the degree of fever. Contrary to the effects with syphilitic patients treated with malaria, none of those with polysclerosis suffered circulatory or renal disturbance. As in other high fever diseases the malaria patients showed slight febrile albuminuria. Swelling of the spleen at times caused pain, and there was occasional slight edema of the liver. Liquid diet was used during the fever period, while an easily digested light diet was used when fever was not present. For the so-called suppression or control of the fever, quinine was used. It is advisable to test the patient for quinine idiosyncrasy, which may occur in perhaps one out of every thousand persons.

All of the authors' patients recovered from the malaria remarkably quickly. In from one to two days, the appetite returned and in less than twenty-four hours heart medication was discontinued. In from two to four weeks hemoglobin and erythrocytes were normal. No doubt the injection of neoarsphenamine after the fever was a material factor in the regeneration of blood. Residuals such as neuralgia, which are frequently noted in the anopheles malaria, did not occur, nor was there any recurring malaria.

Twelve patients were treated with malaria, and as with those inoculated with typhoid vaccine, they were not selected but were given the treatment as they came, even those with chronic progressive sclerosis and total or nearly total disuse of the limbs. All patients were followed up for from two to six months after dismissal, and none were any worse for having been treated with malaria.

The ages of the patients (eight women and four men) varied from 20 to 49. In two cases the disease was of three years' standing, in six from four to six years and in four from eight to eleven years — comparatively old cases. In spite of the poor prognosis of most of the cases the result of the malaria treatment was better than that with typhoid vaccine.

Only one of the twelve cases remained unchanged. This patient, aged 49, with a chronic progressive form of the disease, which had lasted nine years, for one year had not been able to walk. Nine patients grew distinctly better and two very much better. In one patient, for instance, the paresthesia disappeared, and he was again able to feed himself. In another patient the dizziness was relieved and the bladder and rectal symptoms materially lessened. Others were able to write and comb their hair after years of inability to do so. The best results were obtained in the gait. Two patients who had used two walking sticks for months were again able to climb stairs and go for a short distance without their canes.

Only one of the nine patients who were distinctly better grew a little worse again after three months. The remaining eight remained improved for months, and some have even made further improvement. The improvement usually took place immediately after treatment, but occasionally only a month or two later. It is noteworthy that, in the two cases of recurring type, a relapse was suffered; however, the patient responded quickly to added treatment.

Contrary to the observations made in Vienna, the authors have found the malaria treatment superior to the typhoid vaccine. However, they are frank to admit that little is known of the future of these patients. The two who had relapses after several months show that the malaria treatment is not the

last word in the cure of multiple sclerosis. On the other hand, the results in the chronic progressive cases encourage one to go on with these investigations. It may be of benefit to continue to treat the patients with arsphenamine and arsenic after the malaria cure in order to avert a recurrence.

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SYMPOSIUM ON SEXUALITY (I). J. A. HADFIELD, Brit. J. M. Psychol. **5**:161-174 (Dec. 19) 1925.

A symposium on the conception of sexuality was held before the Medical Section of the British Psychological Society on March 25, 1925. Hadfield, who represents the conservative point of view, and whose paper opened the conference, defines its purpose as "trying to discover the scope and meaning of the term 'sexual.'" His main thesis is based on his definition of sexual as "that group of impulsive tendencies whose natural aim is reproduction." He accepts as sexual the normal act of reproduction, any direct stimulation of a sex organ, the various sex perversions, and some forms of self-display. He eliminates with this definition most of the infantile sensory stimulation such as thumb-sucking, urination, etc., and sees in such tendencies as curiosity, observationism, sadism and masochism components which are primarily egoistic and only secondarily utilized for sexual aims. In sadism, for example, when the self-assertive impulse predominates, the primary end is self-preservative, and not reproductive. Early in life these aggressive tendencies are utilized for getting food. Later they are directed toward sexually loved objects and arouse impulses whose "natural end is reproduction." Then the components of sadism are properly included under sexual. Similarly, in masochism there are different components. The submissive impulse is traced to three sources: (1) submission of child to parents; (2) subimssion of the individual to the herd; and (3) submission of the female to the male. This last is distinctively sexual.

In the field of infantile activity, he finds himself most at variance with the teachings of Freud and the psychanalysts. They extend the term sexual to apply to all forms of infantile sensuous gratification, and use the terms sexual excitement and sensuous gratification interchangeably. Five reasons are advanced by the freudians to support this point of view: (1) analysis of adult sexual disorders leads back to these infantile pleasurable activities; (2) stimulation of pregenital zones in children produces genital sensations; (3) the activities of the pregenital zones are organized and later subordinated to the genital zones; (4) persistence of these activities in perversions; (5) the same common feeling characterizes both infantile activities and definitely adult sexual activities, and it is difficult to say when the latter begin.

All these arguments except the first are regarded as inadequate. There exists strong clinical evidence connecting adult sex experiences with these infantile activities. Nevertheless, it is a complete misinterpretation of these biologic functions to call them sexual. The pleasurable element associated with these activities encourages the formation of habits and persistence of the activity, and the center of gratification shifts from one zone to another as one physiologic activity emerges from another. There is no justification for saying they are "organized" and then "subordinated." In abnormal cases these habits persist and become associated with sexual impulses. This secondary abnormal association with sex activities leads to the false assumption that these infantile activities are primarily sexual.

There should be a distinction between the terms sensuous and sexual. Sensuous applies to all forms of physical pleasure regardless of whether there is an end or whether it is indulged in only for gratification. Sexual is retained for those activities whose natural end is reproduction.

THE CONCEPTION OF SEXUALITY (II). JAMES GLOVER, Brit. J. M. Psychol. **5**:175-188 (Dec. 19) 1925.

Glover, in the second contribution, presents the strictly freudian point of view. Rigid definitions are regarded as unimportant and misleading. The real dispute on this subject is on the facts, and not on the definitions. He rejects Hadfield's limitation of sexual as including only that group of impulses whose natural end is reproduction. This insists solely on a teleologic criterion. He could accept it if the natural end were coitus. There is no more justification for regarding reproduction as the natural end of the sex instinct than there is for calling anabolism the end of the nutrition instinct. Freud lays the emphasis on the psychologic characteristics of the impulses in question. He does not deny that there is an ego-component in self display, but insists that there is an admixture with the sexual component.

The perversions are regarded as sexual in themselves and not because these activities arouse genital excitation whose "natural end is reproduction." The pleasure derived by the pervert from his activities is "identical" with the pleasure derived from coitus.

Glover regards Hadfield's conception of sexuality as more comprehensive than Freud's because he limits it to genital activities resulting in reproduction and extends it rather loosely "to include events which, by stimulating genital activities, subserve reproduction." He challenges Hadfield's statement that a child's interest in urination and defecation is not essentially different from interest in food and strangers. He states that very early the child reacts to these activities in the same way as the adult reacts to direct sexual activities, i. e., with shame and guilt. These attitudes are derived partly from the reflected external attitudes of parents to these tabooed interests, but he "feels that the child meets this external pressure with inherited readiness."

On the question of sadism and masochism there is rather a gap between Freud's and Hadfield's points of view. Freud feels that there is a very early sexual component to aggression — that these impulses acquire libidinal significance early in childhood. Hadfield, however, recognizes no sexual component in aggression until it is directly aimed toward sexually loved objects.

Glover feels that Hadfield's insistence on not regarding as sexual the numerous sensual satisfactions found in childhood is tantamount to maintaining that the sexual instinct develops without a background when maturity is reached. He subscribes to Freud's view that these early activities are precursors of adult sexual experience. The most important evidence supporting this point of view is provided by the phenomenon of regression which shows the continuous path of development and that these early infantile tendencies can replace the adult tendencies. The sexual instinct has a genetic background reaching back into early childhood, and evidence for the continuity of normal psychosexual development comes from these sources: (1) observation of children; (2) child analysis; (3) analysis of normal adults. If we fail to accept this genetic view of sexual development, then we must limit our concept of sexuality to adult genital impulses capable of resulting in reproduction. Grouping all infantile tendencies as sensuous, as Hadfield insists, and denying their relationships to later sexual tendencies in the adult is equivalent to denying this continuous genetic point of view.

CONCEPTION OF SEXUALITY (IV). JAMES GLOVER (reply), Brit. J. M. Psychol. **5**:196-207 (Dec. 19) 1925.

Shand's contention that sexual tendencies cannot be classed as such until they are "consciously experienced by all normal persons" would eliminate the sexual instinct from a great group of people (mostly women) who, through cultural influences and training, have brought about an apparent disappearance of sex desires. If one admits that such influences can mask the instinctive life of man, then the objections advanced by Mr. Shand would be validated — and accepting the fact that modification of the instinctive life can be brought about by developmental influences, then Dr. Hadfield's biologic criterion which emphasizes "reproduction the natural end," would be misleading in the study of the sex life, and Mr. Shand's "questionnaire" method would fail to elicit the facts. This method fails to take into account the possibility of certain components of the sex life being repressed.

The criticism which is aimed at his assertion that "there is a psychological identity discoverable in processes grouped as sexual" is based on a misunderstanding. They are not identical, but there is a libidinal quality invested in each — and the difference is in intensity rather than in quality. In the example of oral and genital sensations cited by Shand, the mouth excitation is certainly not identical with the genital sensations. But in the sensations of both there is a libidinal component which is derived from the sexual instinct.

Shand's denial of libidinal excitation in erogenic zones on their own account is denying the essence of Freud's theory of libido organization, which holds that in all normal persons the activities of pregenital zones become subordinated to the genital activity — when once established excitation of one of these zones tends to set up genital excitation which then dominates but does not completely replace sensation in other zones. In some cases, especially in women, when genital sensations have been repressed, the oral sensations which are recognized as sexual occur without any genital excitation. As the primacy of the genital region becomes established the investment of libido in the other zones becomes weaker and is stimulated to the point of conscious recognition only under a strong stimulus. In some cases, especially in women, zones other than the genital may retain a strong libidinal component and stimulation may occur without any genital excitation. When this occurs it is usually the result of repressive cultural influences. A case is cited in which an adult woman was anesthetic to the normal sex act but responded with strong excitement to anal coitus, and there was no accompanying genital excitation. The childhood influences responsible for this condition were the frequent administrations of rectal enemas by a neurotic mother, which were accompanied by considerable pleasure. There were frequent episodes of anal masturbation during the latency period. In this case a rigid biologic criterion advocated by Hadfield would have excluded this activity as sexual. Calling this case abnormal would not dispose of the facts of the case. Such cases would be common if it were not for the strong cultural repressions and resistances against conscious recognition of anal eroticism. In this particular case, stimuli were too frequent and too strong to be repressed and the ego made an adjustment to them.

Freud has not given a formal and fixed definition of sexuality, but has contributed his libido theory which "intelligently strings together developmental data and accounts for such impressive facts as that of displacement, which constitutes the outstanding feature of man's mastery of his sexual impulses and that of regression following his all too frequent failure."

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ON A CASE OF ORGANIC SPINAL HEMIANESTHESIA SHOWING THE PERSISTENCE OF A PECULIAR FORM OF SENSATION AND THE OCCURRENCE OF PHENOMENA OF ALLO-ESTHESIA AND HETERESTHESIA. R. M. STEWART, J. Neurol. & Psychopath. **5**:289 (Feb.) 1925.

The nerve impulses for the different forms of sensation travel in spinal cord tracts, some of which are crossed and some uncrossed. No single afferent fiber in the cord conveys impulses upward from both sides of the body. Complete hemianesthesia, confined strictly to one side of the body, can occur only when all the afferent tracts which convey the impulses from that side of the body are severed, and these only. The lesion must be one that affects only part of each lateral half of the spinal cord, and the parts affected must be dissimilar on the two sides and in a manner complementary. Naturally, such a selective lesion is rare when accident brings about the injury. In such circumstances, if each of the elements of sensation is carefully investigated and found to be absent, does anything remain?

The case presented is that of a young soldier who, on diving into shallow water but not striking bottom, experienced a sudden pain in the neck and at once lost power in his limbs. He was pulled out of the water and soon regained some power in the left upper and lower limbs. Preliminary examination showed: abolition of knee and ankle reflexes on both sides; sensory disturbance to pain and tactile stimuli on certain parts of the right upper limb, on the left upper and lower limbs and on the whole left side of the body below the level of the second rib; narrowing of the right palpebral fissure with contracted pupil and enophthalmos. A roentgenogram on the eighteenth day showed a fracture-dislocation of the fifth and sixth cervical vertebrae.

Details are given concerning the motor weakness which predominated on the right side and the sensory loss which predominated on the left side. Except in certain regions of the right upper limb, there was no sensory change on the right side of the body at any stage of the condition. On the left side, profound sensory changes of an unusual character were found on the trunk and lower limb. The patient replied to all forms of stimuli, but the sensations evoked by the different forms of stimulation were all described as similar. Tactile, pressure, thermal and pain stimuli were not recognized as such, but were described as "queer" or "funny," "like electricity" or as making the patient laugh.

As time went on, further changes were demonstrated. The sensory changes in the right upper limb slowly decreased in severity. After five months the area of hypesthesia in the right arm corresponded to the seventh and eighth cervical and the first and second thoracic roots. In the left arm at the end of two months, no sensory disturbance could be demonstrated. Motor power gradually improved on the right side. Joint sensibility on the left half of the trunk and the left lower limb, however, showed progressive loss. A small area on the left foot, at first responding to compass tests, light touch and pain, soon showed loss of sense to these stimuli. Localization, at first only slightly impaired, also became lost in the left lower limb for all types of stimuli. The sensory functions of the right side of the trunk and the right lower limb remained normal.

An attempt was made to determine the site and nature of the lesion. Extramedullary hemorrhage was practically excluded by the spinal fluid examination. It seems more than likely that the curious complex of symptoms owed its origin to a number of intramedullary hemorrhages in certain localities of the cervical cord. Regarding the vertical extent of the lesion, the integrity of the diaphragm

and the trapezius muscles indicated the escape of the fourth cervical segment and corresponding nerve roots. Clinical observations indicated involvement of the fifth, sixth, seventh and eighth cervical and the first thoracic segments on both sides. The evidence pointed to greater injury to the posterior roots on the right side. It is evident that the lesion was far from a complete transverse one. It is necessary to assume that at full development of the symptoms there was complete involvement of the left posterior columns and the right spinothalamic tract with slight implication of the right corticospinal tract. The change from partial to total involvement of the left posterior columns may have come through a spreading gliosis or a gradual vascular and lymphatic occlusion.

Although the patient could not distinguish between different stimuli, he was yet not completely insensitive to any one of them. The quality of different sensations had disappeared; the sensations had no variation in intensity, extensity or local signature; but sensation of a peculiar form was still evoked. The patient volunteered the statement that it was unlike anything that he had previously experienced. For convenience this condition may be termed "arch-esthesia." This "funny feeling" was evoked by stimuli applied to any part of the left side of the body below the second rib. At the upper and mesial borders were areas in which both ordinary sensations and "funny feeling" could be evoked. He was unable to distinguish two synchronous stimuli from a single one no matter how far apart they were applied. He was also unable to distinguish two successive stimuli if the interval of time that elapsed between them did not exceed four or five seconds. The reaction time to tactile, pressure, pain, faradic and thermal stimuli was on an average about three or four seconds. Finally, on the sole of the left foot there was less disturbance of sensation than in other parts of the affected area.

With the eyes open, the patient had good coordination in the left leg. With eyes shut there were marked difficulties.

"Allocheiria" (Obersteiner) is a condition in which, although sensibility is retained, the patient is frequently in doubt as to which side of the body has been touched. "Allo-esthesia" (E. Jones) exists when, with some sensory defect, the patient is similarly in doubt. In this case, touch stimuli on the affected side were often referred to the opposite side of the body. The condition was therefore one of allo-esthesia.

In final form, a weak stimulus evoked "funny feeling" which had no definite locus, but if its strength was gradually increased, at a certain point the "funny feeling" appeared to jump over to the opposite side of the body, and was referred to a situation that was found to be the accurate mirror localization of the site of the stimuli.

"Heteresthesia" is a term proposed to describe a state in which difference in the degree of sensation is experienced when stimuli of equal value are applied to different parts of the skin. This was demonstrated by drawing a weak faradic stimulus down imaginary parallel lines on the affected side of the body. At certain points only would "allo-esthesia" appear. When these points were joined together they formed lines which corresponded exactly with the boundaries of the radicular or segmental areas.

The possibility of an hysterical condition was thoroughly considered and ruled out in various ways.

It is concluded that the condition seemed to have destroyed all afferent paths between the left side of the body and the cerebral mechanism without injuring those between the left side and the rubrocerebellar apparatus. It is conceivable that the impulses conveying the "funny feeling" ascended in the

spinocerebellar tracts, eventually reaching the cerebrum by cerebellocortical paths. Normally perhaps, the innumerable impulses reaching the cerebral cortex swamp out of consciousness those impulses coming by the circuitous path. The motor phenomena of the left side would harmonize with this assumption. The lines of "heteresthesia" being half as numerous as would be expected for the limits of root areas, it is concluded that there were lines of segmental areas and that overlapping in them does not occur to the same extent.

FAVILL, Chicago.

THE PRACTICAL VALUE OF PSYCHANALYSIS. RICHARD TRAUGOTT, Med. Klin. **22**:49 (Jan.) 1926.

Although psychanalysis has been practiced for about thirty years, the general practitioner and even the neurologist and psychiatrist have only a hazy idea of what it really is and how to apply it. Traugott compares psychanalysis to confession; they are related to each other, the object of both being to still the tormenting unrest of the soul and to reestablish its equilibrium. Each psychanalyst, of course, has his own method. One gets his patient comfortably seated on a divan and asks him to relate his experiences and to give a history of diseases and domestic status. The patient unknowingly is soon in deep water in which he has not ventured for a long time and gradually the little ship of his thoughts loses itself — thoughts come back to him that have long been forgotten; latent memories are revived and soon overstep the threshold of consciousness. Another enters into the dream life of the patient and endeavors to get him to interpret his own dreams; then again there are associations ad infinitum. Still another dwells almost entirely on the dream life, has the patient tell his dreams and from the story and the dreams endeavors to figure out the complex. The latter method undoubtedly is preferable as our true character, longings, desires, hopes and fears are brought out in dreams much more primitively and honestly than in conscious thoughts. Dreams are composed of plastic imaginations and pictures which, as in waking thoughts, simply follow and merge into one another — hence, the frequent transformations, amalgamations and condensations of dreams.

The so-called regression in dreams, as well as in waking impressions, plays an important rôle in psychanalysis. The regression, that is the dependence of imagination not only on experiences and ambitions of youth and early childhood (ontogenetic regression) but also on race and civilization (phylogenetic regression), is no doubt of great importance in the constellation of the actual state of the mind, and frequently is the means of explaining apparent sexual perversions and especially antisocial and criminal tendencies.

The study of the regressions seems to be of the greatest theoretical interest and at the same time of eminent practical value. The dream picture is taken as symbolic, as even our dreams are hampered by conventionality and custom which have become second nature. For instance, a woman, rightfully, and in a perfectly normal manner, desires to be the sexual object of a man; in her dream she is pursued by a man, but critic and convention do not permit her desire to be so easily gratified, and the man appears in the form of a robber. The robber in this case is a symbol of the lover; frequently, dream pictures are symbols of their opposites. However, to compile a list of symbols and attribute to each a certain definite meaning, as has been done by various freudians, is not justifiable. The pursuing robber may have a different meaning and not be the contrary symbol of a lover — all depends on the patient's mental state. This also holds true for the impressions received from the voluntary

telling of the dreams. It is well here to keep in mind that many of the patients being anayzed are hysterical and tend to prevaricate; they are often reticent, and thus confuse the examiner. For example, the author had a patient, a young girl who had a fear complex, with whom he had worked for more than a year without apparent result. Suddenly the author became ill and told the patient that he had to undergo an operation. Two days before the operation the patient asked him if she could be punished for an offense she had committed a long time before. On assurance that she had no cause for worry, she promptly grew better and became practically well.

The examiner must look on everything that the patient tells him as important. The most important work, however, is to be done when the complex has been figured out. Most complexes are based on juvenile experiences; their unimportance must be explained to the patient, and he must be made to understand just what has subconsciously caused his trouble. The following may serve as an illustration: In falling asleep at night one suddenly thinks of an acquaintance met the previous year at the seashore, but the name cannot be recalled. This causes great worry, although there are no unpleasant associations in connection with the acquaintance. The next morning the name suddenly comes back to mind and the worry instantly ceases. Whether this complex theory is correct or not, if the suggestion is properly carried out it seldom fails to bring results.

If a successful analysis has been made, synthesis is introduced and psychotherapy, persuasion, suggestion, etc., may be tried in order to bring about a cure. Not only must the accompanying disease affects be overcome, but more especially the mental pitch of the neurotic person, and his feeling of insufficiency must be influenced. It is the mental attitude in conjunction with a feeling of sexual guilt that leads to fears and phobias and even to all sorts of compulsions, even as the religious feeling of guilt finds an expression in religious ceremonials. Setting new goals, establishing new modes of living, remodeling thoughts etc., are the supreme task of the psychanalyst after an analysis has been made if results are to be obtained.

The psychanalyst need not look on everything the patient tells him as pure gold; he may even assume the opposite meaning ,just as a conscientious confessor is not content to remain on the surface but asks questions in order to get to the depths of the soul, and the success of the confessor rests on the intelligence of his questions.

The confessor and the psychanalyst alike must determine at which point the person confessing falters and what is particularly painful to him, and by questions tactfully put must gradually draw him out. After this has been done and the trouble determined the real task is at hand, as it is not enough to know the condition of the soul; if help is to be obtained synthesis must follow. The confessor must arouse a feeling of contrition and give comfort by the administration of absolution. Then only does the person confessing go away relieved. The parallel drawn between confession and psychanalysis should illustrate the therapeutic value of confidence in a higher power, which is found in religion. Even the most radically materialistic physician should sacrifice his intelligence for the benefit of his patient, and should encourage him in his religion if he is truly religious. Dejerine's statement that a truly religious person cannot be nervous or hysterical contains a certain amount of truth, especially if the religion is not shrouded in mysticism.

In both confession and psychanalysis the task includes a thorough examination or analysis of the conscience and readjustment of the troubled soul. Whether this is brought about by freudian or other technic is immaterial. Although

freudian theories as well as the technical practice of freudianism will no doubt endure, the freudian school generalizes entirely too much, for instance, in that it holds that unhealthy fears, without exception, are due to a sex complex; that hysteria has its origin in infantile sex trauma; that the displacement of complexes leads to the physical symptoms of hysteria; that every dream represents a desire; that the neuroses arise out of typical complexes such as the Oedipus complex, the castration complex, etc. Nevertheless, we are grateful to Freud for more forcibly directing attention to the associations and peculiarities of the mind. There is no sanctified method of psychanalysis or psychotherapy, and no doubt there never will be. The complexity of human individualities entails complexity of psychanalytic and psychosynthetic methods which must be used together to supplement and help one another.

MOERSCH, Rochester, Minn.

THE HEREDITARY DISPOSITION TO SENILE DEMENTIA. F. MEGGENDORFER, Ztschr. f. d. ges. Neurol. u. Psychiat. **101**:387 (Feb.) 1926.

This study was carried out in cases of senile dementia diagnosed clinically and pathologically, the microscopic examinations being carried out by Jakob. None of the cases showed signs of arteriosclerosis. Sixty cases were thoroughly investigated from the standpoint of family history, and from these studies Meggendorfer concludes that there is a familial incidence in senile dementia. In sixteen families, it occurred more than once: eight times in sisters, once in stepsisters, and ten times in parents and children, occurring at times in three successive generations. Meggendorfer reports five families in which senile dementia occurred in two or three successive generations, and believes therefore that this is a dominant characteristic or manifestation of a simple anlage. The objection that the direct transmission of senile dementia occurs only exceptionally (in five of sixty cases) is not important for the reason that since senile dementia is a disease of old age it cannot be traced often in bearers of the tendency who do not reach old age, while the occurrence of senile dementia in younger generations is difficult to ascertain. The assumption, moreover, of a simple dominant characteristic in senile dementia has a certain parallel in another progressive disease, Huntington's chorea, which shows the presence of such a dominant characteristic. The two conditions have another thing in common in that patients with Huntington's chorea occurring in late life show all kinds of psychopathic traits in early life, just as in cases of senile dementia a psychopathic personality can often be traced in early life. While stressing the dominance of this tendency to senile dementia, Meggendorfer states that parents with senile dementia do have children without this malady, and cites five family charts to show this. To explain this one must assume the presence of a recessive characteristic, as, for example, in the case of a family with two parents showing senile dementia and with two sons who were old but without dementia. Such a recessive characteristic is found in brain atrophy. For example, the arrival at a certain age has been shown by Pearson, von Lindheim, Bell and Plötz to be dependent on certain far-reaching tendencies. Recently, Pearl has shown by his experiments on *Drosophila* that different strains of the animal in the same surroundings have different life ages, some being long-lived and others short-lived. In crossing these different types, Pearl obtained only long-lived individuals in the first generation, but in the second generation he obtained both long-lived and short-lived animals. Meggendorfer found the tendency to long-lived individuals a dominant char-

acteristic over shorter life. The recessive anlage of shorter life is dependent on less efficient organs and weakness in defense mechanisms in the individual. With the progress of life to its normal end, certain regressive events occur in which the brain takes a very important part. In how far autointoxication plays a rôle can only be surmised. According to another theory of old age (Ribbert, von Hausemann), the cause of senility lies in the brain.

The question, however, arises, if brain atrophy is a recessive characteristic, whether senile brain atrophy signifies senile dementia per se. The answer to this depends on our conception of senile dementia. If one considers age a physiologic process with regressive changes in all organs, particularly in the brain, then one must concede the truth of this assumption, and look on physiologic brain weakness as the dominant anlage for long life. It is not sharply differentiated from senile dementia for, as Alzheimer showed, senile dementia is really only a pathologic accentuation of the normal senile process. With this pathologic change in the brain, a study of the families shows certain other characteristics which make for senile dementia. For example, many of the patients showed nervous, irritable, irresponsible, schizoid personalities, and indeed the literature contains many statements as to the relationship of senile dementia to dementia praecox. Berze, for example, found that many so-called cases of senile dementia are in reality but late manifestations of a dementia praecox anlage. He calls attention also to schizophrenic patients with a symptomatology similar to senile dementia. Kraepelin remarked on the great similarity of senile dementia to dementia praecox. Bleuler went a step further in his study of senile persons with catatonic symptoms; he concluded that in such cases we really have latent schizophrenia, which first becomes manifest through brain atrophy. Lenz further states that people who develop mental disturbances in later life were never normal in earlier life. From the standpoint of the relation of dementia senilis and dementia praecox, Meggendorfer divides his cases into four groups: (1) Five cases which could be called schizophrenia with development in senile dementia. These occurred in the fifth or sixth decade and showed signs of senile dementia and cortical atrophy on necropsy. (2) Twenty cases with a more or less schizophrenic picture. The average age at the time of occurrence was 70. (3) Six cases in which, in the beginning at least, the patients were anxious and depressed. The average age was 64. (4) Twenty-nine cases showing a simple, uncomplicated dementia or a presbyophrenic picture with disorientation and confusion. The average age was 71. Moreover, about 75 per cent of Meggendorfer's patients were nervous or peculiar in early life. About 40 per cent had personalities that would be described as schizoid psychopathic.

Concerning the relation of senile dementia to dementia praecox, there are two outstanding possibilities: (1) "Senile dementia is possibly only a senile schizophrenia." The possibilities in such a case are that the schizophrenia after a long latency becomes manifest in the senium, or that the senium occurs earlier in schizophrenic persons than in others. For the majority of cases the objection of Kehrera probably holds, that the symptomatology of the two conditions is different, and that the occurrence of latent dementia praecox takes place in the senium only in those cases in which senility occurs on schizophrenic soil. (2) Both conditions, dementia senilis and schizophrenia (dementia praecox), have certain fundamentals in common in the form of hereditary tendencies. Rüdin and Hoffmann believe that dementia praecox is dependent on a dihybrid-recessive modus. Meggendorfer found in his material sixteen cases of true dementia praecox and fifty-eight schizoid personalities. Among

234 children he found 2.1 per cent schizophrenic and 10.7 per cent schizoid personalities. Hoffmann found in the children of schizophrenic persons about 9 per cent schizophrenic and 49 per cent schizoid. Schneider in a similar study found 21.8 per cent schizoid, 22.3 per cent with other psychopathies and 55.9 per cent healthy. Meggendorfer found among 450 blood relatives of senile dementia patients 7.5 per cent schizoid, 25.5 per cent psychopathic and neuropathic, and 67 per cent healthy. The difference is so great that Meggendorfer concluded that he could not agree as to the fact that senile dementia is only a modified dementia praecox. Meggendorfer found other forms of psychopathy and neuropathy more common in his study of senile dementia than the schizoid personality, and found no great frequency of dementia praecox among the children of senile dements.

To answer the question as to whether a weak, less resistive anlage of the nervous system was not the real basis of senile dementia, Meggendorfer compared the families of senile dements with those of forty persons who had cerebral arteriosclerosis, and found a greater incidence of involvement of the nervous system in the families of the former than in those of the latter. He decided that there was a constitutional weakness of the nervous system in the former.

ALPERS, Philadelphia.

PERIARTERIAL SYMPATHECTOMY. R. W. MCNEALY, J. A. M. A. **86**:1918 (June 26) 1926.

Jaboulay, in 1899, was the first to perform periarterial sympathectomy when he removed the periarterial tissues from the femoral artery for the cure of perforating ulcers of the foot. The operation lapsed into obscurity until 1913, when Leriche's article concerning the removal and section of perivascular nerves for the relief of painful syndromes of arterial origin and in some trophic disturbances appeared. In 1921, Leriche described the effects of the operation, reviewed his observations and researches and reported the results in sixty-four cases. He has suggested its use in Raynaud's disease, erythromelalgia, causalgia, trophedema, trophic ulcers, painful stumps, ischemic sequelae, varicose ulcers, varicose eczema, intermittent claudication, thromboangiitis obliterans and disorders of the Babinski-Froment type. Ununited fractures, acute infections of the extremities, tuberculous bone lesions, leprosy, osteomyelitis and female pelvic disturbances are being so treated now. Anatomically, the field covers every approachable vessel in which denudation will afford relief to local or adjacent pathologic conditions.

There have been so many publications dealing with the anatomy, physiology, case reports and modifications of technic and with such widely varying results that McNealy feels it timely to review the field. The operation was first designed for those sympathetic dysfunctions of the vasomotor system which showed a preponderance of vasoconstrictor action; but now it is used in conditions that may be benefited by the resultant hyperemia. The technic of the operation consists in the isolation of a peripheral artery over a distance of from 8 to 10 cm., longitudinal division of the adventitia and then circumferential dissection.

The following reactions occur: (1) a primary contraction at the site of denudation; (2) in from three to fifteen hours, dilatation and hyperemia, which persist from a few days to several weeks. Many modifications have been suggested which are reported to be less radical and yet produce the same reactions.

The primary contraction has been explained as due to excitation by trauma of the vasoconstrictor fibers which were thought to pass from the dorsolumbar outflow of the ganglionated cord as a network in continuity along the vessels of the extremity, thus removing the preponderance of vasoconstrictor tonus. It was also thought that any long sensory paths that might reflexly influence vessel tonus or carry painful sensations could likewise be interrupted.

McNealy believes that these phenomena do not result from interruption of the long vasomotor tracts supplying the peripheral vessels, because the phenomena are frequently transient and because experimental investigation shows that the vasomotor nerves have a segmental arrangement and can be interrupted only locally. He cites the work of Claude Bernard, Kramer and Todd, and Potts and Langley in support of his argument. He then quotes from Bayliss a survey of the factors controlling normal blood vessel tonus, which are: the natural property of smooth muscle to be in a state of tonus, and the continuous constrictor impulses sent out by the tonic excitation of the vasoconstrictor centers. Other factors are the contractions set up by epinephrine hydrochloride in the blood and possibly the contraction in response to the natural stretching force of the blood pressure. McNealy then cites the work of several men. He concludes that we have no proof that either the long vasomotor tracts or the long sensory tracts travel in the adventitia of the blood vessels. There are vasomotor and possibly sensory changes following periarterial sympathectomy, no doubt, but we cannot attribute these to an attack on the fibers coursing in the sheath of the vessels, as experimental work and operative procedure in no way approaching perivascular neurectomy produce similar reactions. It may be that rest in bed, reactive hyperemia of inflammation following local trauma and healing, interference with sensory conductivity, increased physiologic activity of voluntary muscles accompanied by active hyperemia, and psychic effects are the actual factors.

The clinical results have not been satisfactory, perhaps owing to lack of knowledge concerning the exact pathogenesis of many conditions for the relief of which the operation has been performed. One is reminded, states McNealy in reviewing the literature, that the enthusiasm for the operation is not borne out by a critical analysis of case reports, many authors waxing eloquent over the early improvement and appending an apologetic addendum that the apparent cure was only transient. Lumbar sympathectomy seems to have a more logical foundation than a perivascular one, but the gravity of the operative procedure speaks against it. Indeed the periarterial operation, although a comparatively simple procedure, has been attended by many reported complications. McNealy, in conclusion, accepts neither the theory nor practice of periarterial sympathectomy, and believes it should be discarded. He believes that a more careful study of the sympathetic system should be made and particularly of the pathogenesis of various vascular diseases.

CHAMBERS, Syracuse, N. Y.

INTRAVENOUS INJECTIONS OF SALT SOLUTION IN THE TREATMENT OF SCLERODERMA. FRANZ HERZOG, Med. Klin. **22**:1178 (July 30) 1926.

Because of the good results obtained by intravenous injections of hypertonic salt solution in the treatment of intermittent claudication, the author decided to try this method of treatment in scleroderma. Ten cubic centimeters of a 10 per cent solution was injected into the veins with the following results:

A woman, aged 35, in whom the disease was of fourteen years' standing, in the beginning had as the sole symptom coldness and blueness of the first

joint of the index finger part of the time. This disappeared in the summer and recurred in winter. For two years other fingers on both hands had been similarly affected, and pain and tingling had occurred. In the preceding year the condition had grown worse; the skin hardened about the fingers, became taut and brown, and small, painful sores developed. Gradually the skin about the face, breast and feet became harder and brown, and there was a hardening over both elbows. At the time of examination, the patient could not use her hands and had to be assisted in eating and in dressing herself. Roentgenograms showed atrophy of the phalanges. The face showed typical changes of scleroderma. The erythrocyte count was 5,190,000; the white cell count, 10,400. The albumin content of the blood was normal. The patient had thirty injections of salt solution. Her condition improved greatly; pain decreased, and the sores cleared up. However, owing to poor veins, the treatment could not be continued; she was therefore ordered to take 300 cc. of a 9 per cent solution daily. When she left the clinic she was much improved; she had pain only occasionally in cool weather, and the sores on the tips of the four fingers were better. She continued the dosage at home, and in three weeks the pain had disappeared entirely. The cyanosis and brown color disappeared; the skin grew softer and warm; the movements of the fingers were practically normal, and the patient was able to perform all her housework.

In this article eight cases are described in detail; all of the patients reacted favorably to the salt solution except the last two; these showed atrophic changes to such a degree that the veins could not be recognized and injections were impossible.

The vessel changes in scleroderma are known, but there are various opinions as to their significance. One authority says that scleroderma is the result of disease of the terminal arteries. However, this theory is not logical; it does not explain the characteristic vessel changes of scleroderma, and disturbances of peripheral circulation of long standing, such as occur in arteriosclerosis, never develop. As against this hypothesis is the argument that in scleroderma the degree of alteration of the vessels cannot be compared with those in the tissues; the vessels may be intact when there are decided tissue changes.

Doubtless, however, diseased arteries play a part in the manifestation of numerous other symptoms in scleroderma. Vessel changes, according to Cassirer and Hirschfeld, begin in the adventitia and extend to the walls of the small arteries, causing an increase of connective tissue. There is also an increase of muscle elements and endothelial cells. In some cases the intima shows the greatest change. Later, the vessel wall becomes atrophied and the adventitia disappears completely, yet the disease of the vessels does not compare with the changes of the surrounding tissue. The tissue surrounding diseased vessels may remain unchanged and vice versa.

Although the characteristic tissue changes of scleroderma cannot be ascribed to vessel changes, it is nevertheless probable that disease of the vessels is important in the production of symptoms dependent on disturbance of the peripheral circulation. In the beginning of the disease vasomotor disturbances, local syncope and asphyxia, and even the picture of Raynaud's disease appear. These symptoms may disappear, but in some cases they continue after the development of the scleroderma, so that the two disease pictures merge into one another. The vasomotor disturbances may be due to anatomic changes in the vessels. Spasms often occur in diseased arteries, as may be verified in patients with intermittent claudication. The disease and disturbance of function of small arteries may play a rôle in the early edema and pigmentation.

The injection of hypertonic salt solution in the author's patients first relieved the skin symptoms; then the pain disappeared, and finally the cyanosis and pigmentation. The sclerodermic tissue improved later and to a less degree. Although this treatment offers merely an improvement and not a complete cure, the author recommends it because of the results he has obtained, and especially because other methods of treatment are even less reliable. Results may be looked for chiefly in cases dominated by vascular symptoms, in which the tissue is not indurated and atrophied.

MOERSCH, Rochester, Minn.

PHYSIOLOGY OF THE SYMPHATHETIC NERVOUS SYSTEM IN RELATION TO CERTAIN SURGICAL PROBLEMS. ALEXANDER FORBES and STANLEY COBB, J. A. M. A. **86**:1884 (June 19) 1926.

The proposal to relieve spastic paralysis by surgical interruption of the sympathetic innervation of the muscles involved originates in the theory that so-called "plastic tonus" depends on the sympathetic nerve fibers and that the abolition of this tonus will relieve the pathologic condition. Bocke established an apparent innervation of skeletal muscles by sympathetic as well as by cerebrospinal nerve fibers, although it is still unsettled whether these sympathetic fibers exist in all muscles and whether there is any histologic difference in muscles so innervated and those not supplied in this way. Some workers report loss of tonus in skeletal muscles following section of the sympathetic nerve, while others fail to substantiate this. Sherrington called attention to the remarkable plasticity of the limb of a decerebrate animal, attributing it to proprioceptive reflexes but ascribing no particular neuromuscular mechanism to the phenomenon. Langelaan sought to establish a contractile and a plastic mechanism in muscle function, and Hunter attempted to show that sympathectomy eliminates the plastic element, but that the contractile element remains undisturbed.

Forbes and Cobb believe that Langelaan's hypothesis is based on misunderstanding of the work of previous observers, and also that it does not harmonize with nerve-muscle function — the all-or-none response of muscle to an adequate stimulus by which all the known parts of plastic tonus can be explained. Artificial stimulation as well as drug action fails to produce contractile responses of the muscles innervated by sympathetic fibers. Previous investigations of sympathectomy tend to show no consistent change in tonus, and Hunter's positive results are too few to justify a final conclusion. Although the theory of separate contractile and plastic functions is untenable, it is nevertheless possible that sympathectomy might have some effect. If the sympathetic fibers participate in decerebrate rigidity, we should expect a decrease in rigidity when they are cut. If the contraction of fibers is more sluggish than that of fibers innervated from the central nervous system, a more brisk reaction should result from section.

With these facts in mind Forbes, Cannon, O'Connor, Hopkins and Miller conducted experiments on seventeen cats. In eleven the forelimb of one side was deprived of its sympathetic innervation by removal of the stellate ganglion, while in the other six the abdominal sympathetic chain was removed on one side. In three, decerebration was performed first. In the other fourteen, sympathectomy was followed in from three to eighty days by decerebration. The technic is described and curves are shown. The conclusion is that any loss of tonus in consequence of the operation is insignificant and that decerebrate rigidity is undiminished after sympathectomy, while the plastic features continue.

A later contribution by Kuntz and Kerper on the otherwise intact animal with a more refined mechanical method seems to show a consistent diminution of tonus after sympathectomy, although we must remember that the intact cerebrum here complicates the interpretation of the results. The work of the same men on birds seems to show the highly suggestive preponderance of a more plastic postural reflex mechanism in the sympathetic than in the somatic nerves — the somatic nerves tending to hold the wing in its normal posture but being inadequate to do so without the sympathetic nerves; the latter tend to make the wing retain whatever posture is imposed on it.

Obili reports that only with the onset of fatigue does sympathetic stimulation increase the contraction evoked by somatic stimulation. The sympathetic effects show a much longer latency. These observations suggest a difference in the action on the muscle of the somatic and sympathetic nerve impulses, perhaps a chemical change in the state of the muscle and a yet to be explained mechanism which counteracts the tendency to fatigue. At any rate, there are many factors that cause confusion.

Forbes and Cobb feel that there is no justification for differentiating a contractile and plastic component in skeletal muscle; that it is probably premature to ascribe successes to the operation of sympathectomy itself and that perhaps incidental features in the treatment, such as exercises, mental suggestion, hospital care and nursing, are responsible for whatever good is accomplished.

CHAMBERS, Syracuse, N. Y.

OBSERVATIONS CONCERNING DELUSIONS OF PERSECUTION AMONG THE BLIND. (PARANOID REACTIONS RESULTING FROM BLINDNESS.) J. SANCHIS BANÚS, Schweiz. Arch. f. Neurol. u. Psychiat. **18**:141-146, 1926.

The writer had the opportunity to make observations on two blind patients, which appeared to illustrate the effect of blindness on the development of persecutory delusions. The first patient was a man who was taken to the hospital following a collision with an automobile; apparently he was not injured. When an attempt was made to assist him after the accident, he became greatly excited. According to the history he gave, he had been blind as the result of trachoma since the age of 5 years. He related that he made his living by begging on the steps of a church in Madrid and that he had collected large sums of money. The practice of begging was soon brought to a stop by some government official, against whom the patient developed a great hatred. He was under the impression that the police were watching him and believed that some one said, "This man must not be let out of our sight." He also heard the heavy steps of two policemen. He remained at home and barricaded his house, until compelled by hunger to venture forth at night in quest of food. On the day of the accident, he had scarcely left the house when the police conspired against him. The noise of the automobiles he regarded as signals. He was struck by a vehicle ordered to do so by the same magistrate who had forbidden the begging, and immediately thereafter was pursued by a crowd of people who delivered him to the place in which he now found himself. He could not be convinced that he was in an asylum, but believed that the other patients pretended to be insane. He was fairly well oriented but lacked all insight.

The second patient was a married woman, aged 32. She had become blind three years before as the result of the explosion of an oil lamp. A friend

had unfortunately told her of improper relations on the part of one of their acquaintances. This upset the patient very much. She suspected other women, including her sister, of intimacy with her husband. The father had been a rather irritable man, and the brother had evidently suffered from an anxiety hysteria. The remark of the patient that a friend "had opened her eyes" seemed rather paradoxic.

The first patient did not recover. The second patient recovered gradually. The diagnosis in both cases was somewhat difficult; however, the writer believes that the blindness was a rather important factor in precipitating the paranoid reactions.

WOLTMAN, Rochester, Minn.

IS INSECT METAMORPHOSIS INFLUENCED BY THYROID FEEDING? STEFAN KOPEĆ, Biol. Bull. **50**:339 (April) 1926.

Thus far no uniformity of results has been noted in investigations on the influence of the thyroid gland on the rate of metamorphosis in insects. Northrop fed larvae of *Drosophila* with thyroid substance and concluded that the drug was not "adequate food" for the animals examined. Kunkel found abbreviation of the pupal stage of the fly *Lucilia*. Romeis and Dobkiewicz found that flies (*Calliphora vomitoria*) fed on thyroid gland underwent pupation later than control specimens. Finally, Kahn found that in *Corethra plumicornis, Ecdyurus forcipula* and *Tenebrio molita* thyroid extract has no influence on the metamorphosis of the larvae.

Kopeć experimented on caterpillars (*Lymantria dispar*) from four lots of eggs derived from four brother-sister matings of moths belonging to the same pedigree. One group was fed willow twigs sprinkled with an aqueous solution of thyroid extract, while a control group was fed willow twigs sprinkled with water. The leaves were administered daily in slight excess of requirements, and no difficulty in feeding was experienced with the thyroid-fed caterpillars.

The results of the experiments may be summarized as follows: (1) Administration of thyroid caused no distinct changes in the larval or pupal period. The only effect was a distinct diminution in the weight of the chrysalids. (2) "Neither the variability of the pupation term, nor that of the emergence of the moths . . . underwent any regular or essential changes as compared with corresponding items of the control materials." (3) Compound solution of iodine (Lugol's solution) had no distinct influence on the rate of pupation of the caterpillars of *Pieris brassicae*, and its only effect was the negative changes in pupal weight. (4) The mortality of specimens fed with thyroid and iodine was no greater than in control specimens.

Despite the negative results obtained in these experiments, Kopeć believes that there is a "hormonal influence exerted by the brain on insect metamorphosis." Positive results have been obtained by some investigators. Nowikoff reports an increase in the rate of multiplication of infusorians in aqueous extracts of thyroid gland. Hankó observed a positive influence of extracts of hypophysis on the rate of molt, regeneration and growth in *Asellus aquaticus*. Wulzen reports that the thyroid gland has a positive influence on the rate of division and growth of *Planaria maculata*, and from the experiments of van Herwerden we know that the cortical substance of the suprarenals of the ox has an accelerating influence on the multiplication of *Daphnia pulex* as wel' as on the growth of this crustacean.

ALPERS, Philadelphia.

THE EFFECT OF SYMPATHECTOMY ON SPASTIC PARALYSIS OF THE EXTREMITIES. LOYAL DAVIS and ALLEN B. KANAVEL, J. A. M. A. **86**:1890 (June 19) 1926.

The authors repeated the work of Royle and Hunter with respect to the influence of sympathetic denervation on the pattern of decerebrate rigidity. The first part of the article is devoted to a brief discussion of the dual innervation of striated muscle.

Eighteen cats were operated on and were observed for from two to nine weeks. No changes in the character of voluntary or reflex movements were observed. Twelve cats were decerebrated by ligation of the basilar artery at a selected level and simultaneous ligation of the common carotids. In all, the onset and maintenance of decerebrate rigidity was exactly similar in all four extremities, including the one previously deprived of its sympathetic innervation. The authors believe that normal muscle tone and the pattern of decerebrate rigidity do not depend on sympathetic innervation.

Hunter and Royle applied their theories of tonus to selected cases of spastic paralysis and suggested sympathetic ramisectomy to relieve these patients of the presence of excessive plastic tone. They considered cases of paralysis agitans, postencephalitic Parkinson's disease and degenerative or traumatic cord lesions as unsuitable, and confined their efforts to cases of Little's disease, traumatic cerebral cortical lesions and vascular cortical lesions. They reported definite improvement in these patients. The authors have performed ramisectomy, and in some cases even have completely divided the sympathetic chains in the cervical or lumbar regions in all the types of cases mentioned above. They conclude with Hunter and Royle that there is no benefit in the cases mentioned as unsuitable, but they also believe that there is no improvement in the other cases; they therefore cannot confirm the work of Hunter and Royle. Concerned in hypertonicity are the corticospinal tract, the extrapyramidal system and the brain stem. Cerebellar cocontraction as well as some peripheral mechanism of muscle metabolism may also play a part. A discussion of the various theories regarding hypertonicity, particularly so-called plastic tonus follows, and Davis and Kanavel believe that not clinical results but only careful physiologic research and objective measurement will lead us to an explanation. They conclude that the sympathetic nervous system may have some function dealing with the metabolism of muscle — probably a chemical function.

CHAMBERS, Syracuse, N. Y.

DIFFERENTIAL WATER ABSORPTION BY CENTRAL NERVOUS SYSTEM TISSUE. J. A. HALDI and J. W. RAUTH, Am. J. Physiol. **75**:294 (Jan.) 1926.

In this paper, a report is made of experiments carried out on rabbits to determine the degree to which water is absorbed by different portions of the higher central nervous system. The parts actually examined were the cerebrum, midbrain, cerebellum and medulla. Study was made not only of simple water absorption but also of its absorption in the presence of sodium chloride sixth molar, glucose sixth molar, hydrochloric acid three ten thousandths normal (3N/10,000) and creatinine (0.00264 Gm. per hundred cubic centimeters). Simple water absorption was found to vary in relation to the specific tissue examined, being greatest in the case of the medulla, somewhat less for the midbrain and cerebellum and least for the cerebrum. In the presence of sodium chloride absorption was definitely diminished, with the cerebellum showing greatest reception and the other tissues much less and in about equal degree. Absorption was also diminished by glucose, cerebral substance absorbing the least,

the remaining tissues incorporating more and in about the same amounts. For hydrochloric acid, observations were made only on cerebral and midbrain material with practically no change as compared to the results found in relation to water alone. This the authors explain as due in all probability to the fact that in the latter instance the acidity produced post mortem induced an approximately equal acid content in the distilled water employed for the test. No change in the absorption curves was found to result on the addition of creatinine.

In general it would appear that the process of water absorption by tissue, probably a colloid chemical phenomenon as suggested by Fischer, depends in great measure on the actual chemical composition of that tissue and is influenced particularly by its acidity. In the light of the knowledge that various parts of the central nervous system vary in chemical composition, the differences noted for the areas studied would seem explainable and especially so in view of the further differences caused by the addition of certain special chemical agents as indicated. This work is of definitely practical interest as bearing on the finer pathophysiology of brain swelling and edema and as shedding some light perhaps on the nature and occurrence of localized or circumscribed forms.

RAPHAEL, Ann Arbor, Mich.

THE USE OF SODOKU IN THE TREATMENT OF GENERAL PARALYSIS: A PRELIMINARY REPORT. H. C. SOLOMON; A. BERK; M. THEILER and C. L. CLAY, Arch. Int. Med. **38**:391 (Sept.) 1926.

The authors present evidence showing the value of fevers in the modification of syphilis of the nervous system and state certain disadvantages in the use of malaria for this purpose, such as the necessity of transmitting the disease from patient to patient, the danger of severe complications, and a certain mortality. They find that in sodoku, or rat-bite fever, these unfavorable possibilities are practically eliminated.

The specific organism of sodoku is *Spirochaeta morsus-muris*. The disease is acquired by the bite of a rat or of several other kinds of animals infected with the disease. A sore suggestive of a chancre appears at the locus of the bite. After from five to fifteen days, fever occurs and skin lesions of various types appear over many parts of the body. The temperature rises to from 104 to 105.5 F., and is of the intermittent type. The disease is self-limited and is readily controlled by arsphenamine.

The organism of sodoku can be kept alive indefinitely in laboratory animals. The material used for human inoculations in cases of general paralysis consists of the citrated blood taken under aseptic precautions from the heart of an infected guinea-pig or rat. The inoculations are made on the anterior surface of the thigh. A sore develops at the site of inoculation, and about three days later attacks of fever occur, the temperature rising to from 102 to 104 or 105 F., remaining up for a few hours or several days and then descending. During the fever, the lymph nodes are enlarged and skin lesions appear. All clinical symptoms disappear with great rapidity following the injection of arsphenamine. By intravenous inoculation, if care is taken not to allow the blood to get into the skin at the point of venipuncture, no primary sore is produced.

The authors feel that the results of the treatment cannot be completely evaluated for several years. They have found that clinical and serologic improvement has taken place in some of the patients suggesting that the method has therapeutic value at least equal to that of malaria, and that in addition it possesses a number of practical advantges.

VONDERAHE, Cincinnati.

STUDIES ON THE CONTROL OF BLOOD PRESSURE WITH HEPATIC EXTRACT. A. A. JAMES; N. B. LAUGHTON and A. B. MACALLUM, Am. J. Physiol. **75**:392 (Jan.) 1926.

It has been demonstrated by a number of workers that extracts obtained from various animal tissues exert a certain lowering effect on blood pressure. Of particular interest in this connection, in the light of recent study, are the extracts derived from liver substance. The present work constitutes one of these more recent investigations bearing on the nature of this suggested depressor property of hepatic tissue. The authors' extract was prepared as follows: The liver mash (in Ringer-Locke solution) was frozen and thawed, following which the protein content was removed by lead acetate (or alcohol). The active principle was then precipitated by phosphotungstic acid, concentrated and purified by ether solution and resolution in saline.

When administered intravenously in rabbits, from 1 to 2 cc. of this extract was found to reduce the hypertension resulting from the injection of 1 cc. of epinephrine (1:10,000) very promptly and consistently to normal and even subnormal levels. Like effects were observed in the hypertension produced by solution of pituitary, iso-amylamine and parahydroxyphenylethylamine. Further, in rabbits untreated with any of these pressor agents, the blood pressure was immediately reduced much below its normal height by small intravenous doses. The depressor effect, it might be added, was found to be prolonged, lasting a number of hours.

As to the actual identity of the pressor principle, the authors at this time have little to offer definitely save that it is ether-soluble, does not give the biuret reaction and is not either histamine, as Abel and Kubota have supposed, or choline. This work, although preliminary, appears of really first importance not only physiologically but also clinically, bearing directly and significantly on the general problem of hypertension, its pathologic concomitants and sequelae.

RAPHAEL, Ann Arbor, Mich.

NOTES ON HYPERPEXIC ACTION (NEUROPEXIA) ON NERVOUS DISEASES. B. DUJARDIN, J. de neurol. et de psychiat. **26**:83 (Feb.) 1926.

Pexic action is the affinity of tissues for foreign bodies and especially for toxins and proteins. A cutaneous territory which is or has been the seat of an inflammation that has disappeared clinically becomes hyperpexic in a general way for toxins and proteins for a long time afterward. This principle of hyperpexia can be applied to lesions of the nervous system. For example, in tabes, even when the humoral and clinical signs are fixed without evidence of being in an active stage, it is possible to provoke a violent attack of pain by injecting small doses of tuberculin. The chronically inflamed roots are hyperpexic for tuberculin.

Applying these theories to general paralysis, it must be remembered that syphilitic meningo-encephalitis may be present without clinical signs of general paralysis. What is the cause that acts, sometimes brutally, and makes the meningo-encephalitis pass from the asymptomatic or silent phase into the one of clinical general paralysis? Is it not the hyperpexia of the inflamed cerebral cortex that renders it more susceptible to common toxins? The kidney, often functionally involved in general paralysis, may be the origin of intoxicating agents, the action of which at the level of the cerebral cortex is strengthened by the condition of hyperpexia. In tabes also intoxications are likely to provoke acute episodes. The evolution of general paralysis is then influenced by two distinct but interdependent factors: (1) fundamental, the diffuse and progressive syphilitic meningo-encephalitis; (2) hyperpexia or neuropexia, which is the

result of the inflammation and which renders the brain cortex hypersensitive to the various toxins.

The theory of hyperpexia or neuropexia illuminates the pathogenesis of the vascular crises in tabes and of postserum neuritis as well as some peculiarities of the evolution of general paralysis.

FERRARO, Washington, D. C.

A CASE OF "EUNUCHOFEMINISIMUS" WITH PARKINSONISM AND PSYCHIC ALTERATIONS FOLLOWING EPIDEMIC ENCEPHALITIS. L. KORST, J. f. Psychol. u. Neurol. **32**:57, 1925.

The author believes that the centers for water, sugar and temperature regulation are placed about the third ventricle: the ganglion paraopticum, the nucleus periventricularis and the tuber cinereum. Disease of these nuclei would therefore produce diabetes insipidus or mellitus, dystrophia adiposogenitalis, acromegaly, dwarfism, etc. The case reported is that of a chauffeur, aged 21, who complained of headache, "hystero-epileptic" attacks and pain in the entire body, coming on one year after an attack of encephalitis. There was gradual increase of weight; loss of the hair of the face and body; atrophy of the testes and impotentia coeundi with retained libido; polydipsia and polyuria, without sugar in the urine; increase of fat of feminine distribution, and well developed breasts. In walking there were no movements of the upper limbs, especially the right. Muscle tonus was increased, but the reflexes were normal. There were no pathologic reflexes. The sella turcica appeared normal. Mentally, the man was irritable and excitable, and he confabulated.

The author separated the clinical picture into three distinct symptom complexes: (1) "eunuchofeminism," shown by decrease in size of the gonads plus the feminine type; (2) polydipsia, polyuria and subnormal temperature; (3) parkinsonism plus psychic abnormalities.

The author believes that polyuria may be hypophysial in origin as well as neurogenic, and dependent on an alteration of the ganglia paraoptica and of the tuber cinereum. The hypothermia and hyperthermia depends on alteration of the tuber cinereum. In view of the normal sella turcica, as shown by the roentgen ray, the author believes that the disease is probably near the third ventricle. To explain the case completely, the author postulates disease in the striate system, as the cause of the parkinsonism, the ganglion paraopticum and the tuber cinereum, and perhaps in various parts of the cortex.

WINKELMAN, Philadelphia.

SYMPATHECTOMY IN ANGINA PECTORIS. ELLIOT C. CUTLER and JACOB FINE, J. A. M. A. **86**:1972 (June 26) 1926.

Cutler and Fine emphasize the importance of correct diagnosis and the elimination of extraneous results if we are to evaluate operative results correctly. A sufficient number of cases has been reported to justify a choice as to the operative technic, and the authors conclude that the Jonnesco procedure (ablation of the stellate ganglion), although a more formidable operation, is preferable to superior cervical sympathectomy. Seven cases are briefly reported, and the authors comment on them as follows: Single or bilateral extirpation of the superior cervical ganglion will frequently give temporary complete or partial relief and often will fail, the complete operation being less likely to fail. A left-sided Jonnesco procedure appears at times to result in a right-sided angina, but this is due to the fact that the residual right-sided angina, not noticed previously by the patient, is now noticed because of the relief of the left angina. Certain patients eventually totally relieved will

still have pain, usually less severe, for a few weeks or months after operation, indicating the insufficiency of our knowledge concerning the sensory innervation of the heart.

No deleterious effects on the cardiac capacity as a result of operation have been observed in any case. Syphilitic patients should benefit from the operation as well as others, provided due consideration has been given to the effects of surgical trauma and general anesthesia. The dangerous cases are those showing advanced cerebral arteriosclerosis combined with severe coronary disease. The patients are made definitely worse by sympathectomy, although there may be some relief of pain. Distressing postoperative complications are the Horner syndrome, pains in the shoulder, neck, face, jaw and arm of the side operated on. The authors favor the partial Jonnesco procedure, unilateral or bilateral according to the case.

CHAMBERS, Syracuse, N. Y.

STUDIES ON EPILEPSY: III. THE INFLUENCE OF INCREASE OR DIMINUTION OF INTRACRANIAL PRESSURE ON THE SUSCEPTIBILITY OF ANIMALS TO CONVULSIVE SEIZURES. C. A. ELSBERG and F. H. PIKE, Am. J. Physiol. **76**:593 (May) 1926.

In this study, alteration of intracranial pressure was induced, in cats, by the intravenous injection of distilled water, the intracranial injection of petrolatum and paraffin (pressure increased) and by the intravenous administration of 5 per cent sodium chloride and 25 per cent glucose (pressure decreased). The authors found that in subjects in which the intracranial pressure had been raised, only from 0.02 to 0.025 cc. of absinthe essence per pound of animal was required for convulsive response, in striking contrast to the usual or "normal convulsing dose," from 0.035 to 0.04 cc. On the other hand, when the intracranial pressure was decreased, the opposite tendency was noted, the required convulsing dose being much greater even as compared with normal animals, from 0.05 to 0.06 cc. Estimations of the total amount of absinthe needed to induce the "first convulsion" and for lethal seizures revealed like contrasts. For normal animals 0.075 cc. was necessary for "first convulsions," 0.047 cc. when the pressure was raised and 0.136 cc. when the tension was decreased. Similarly, the fatal convulsive dose for cats with increased pressure was found to be 0.188 cc. as compared with 0.412 cc., in subjects with lowered pressure. In their discussion the authors offer two especially interesting suggestions: that improvement in epilepsy following starvation may in some measure be due to diminution of intracranial pressure depending on lessened fluid intake, and that jacksonian attacks may conceivably be attributed to localized variations in intracranial tension. These studies of Elsberg and Pike are obviously of practical significance and attest the value of the experimental method in the investigation of this important clinical problem.

RAPHAEL, Ann Arbor, Mich.

THE OPERATIVE TREATMENT OF GASTRIC CRISES. MACDONALD CRITCHLEY and J. M. WOLFSOHN, J. Neurol. & Psychopath. **5**:318 (Feb.) 1925.

The conflicting results of posterior root section and other surgical procedures for the relief of tabetic crises are emphasized and illustrated with five case reports. Evidence for the existence of afferent paths in anterior roots is discussed. Two cases are presented in which anterior and posterior roots were deliberately sectioned. The results were unsatisfactory. Cordotomy, or section of the anterolateral tracts needs further investigation, but we have no proof

that the anterolateral tracts contain the somatic afferent fibers. Occasional successes after very different procedures call for a revision of the pathogenesis of gastric crises. Forster and others have suggested that there may be two chief types of crises. Sympathetic crises may come from irritation of sensory sympathetic fibers of the stomach which reach the cord through certain dorsal roots. Pain is the prominent symptom and vomiting may be absent. Intense hyperesthesia of the skin of the abdomen and exaggerated epigastric reflexes are present. A low hydrochloric acid content is found in the gastric juice. Vagal crises are characterized by intense nausea and vomiting with pain conspicuously absent. A distressing gnawing feeling may be present, but there is no hyperalgesia of the abdominal wall. Hyperchlorhydria is present. This theory would explain some of the hitherto apparently contradictory results obtained. Thomsen states that temporary relief can be afforded by atropine in the vagal crises. Heile advocates epidermal injection of epinephrine hydrochloride for diagnosis, relief suggesting a sympathetic origin. Cases may exist in which both systems are involved. In view of the problems unsolved, a plea is made for thorough and systematic investigation into the nature of these crises by modern laboratory methods.

FAVILL, Chicago.

METHODS OF TESTING THE SENSIBILITY OF TEETH. INVESTIGATION OF THE SENSITIVITY OF HEALTHY DENTINE AND INTACT TEETH TO WARMTH. MAX SPRENG, Schweiz. Arch. f. Neurol. u. Psychiat. **18**:40-56, 1926.

Little work has been done on this subject. It would seem that there may be some fluctuation in sensitivity of the same tooth under varying conditions. The teeth of chlorotic girls, pregnant women and the aged appear to be more sensitive. Steiner, in 1901, found that light touch sensations were not perceived; however, he noted that these subjects were sensitive to pressure. He investigated temperature sensation by heating a thermometer in a sand bath and bringing the bulb in contact with the tooth to be tested. He found that warmth could be appreciated at 80 C. and that pain was produced at 95 C. At 5 cold was felt, but cold produced no pain even at —15. Türkheim questioned whether teeth were sensitive to temperature and said that a temperature of 27 would give rise to a sensation of pain and not heat or cold. He tested carious teeth which had been excavated but were still painless. The question arose whether carious teeth could be regarded as normal, even after a clean cavity had been prepared.

Spreng found that intact teeth and healthy dentine could endure high temperatures. He devised a testing apparatus made by attaching a small strip of copper to a cautery loop, and this, in turn to the bulb of a mercury thermometer. Asbestos insulation was placed between the cautery loop and the thermometer. The writer stated that this investigation would have to be carried much further before any conclusions could be drawn; however, he was of the opinion that his instrument might be useful in the investigation of sensation for warmth in other parts of the body.

WOLTMAN, Rochester, Minn.

ACUTE HALLUCINATORY PSYCHOSIS. G. VERMEYLEN, J. de neurol. et de psychiat. **26**:139 (March) 1926.

The author describes eight cases of acute hallucinatory psychosis in order to sustain the clinical individuality of the condition, as had been claimed by Magnan. The acute hallucinatory psychosis, according to Vermeylen, presents

the following characteristics: 1. It develops on a certain hereditary basis with predominance of emotive conditions. 2. The previous history often reveals a deficient physical condition and always a mental condition characterized by slight intellectual debility and hypermotivity. 3. The disease is recognizable by its sudden onset; by the polymorphism of the symptoms due to psychic excitement; by the eventual concomitant confusion, mania, depression, etc.; by the predominance of delusional states very poorly or not at all systematized; by various hallucinations, and finally by its brief duration.

Up to the present, the author considers these paroxysms of delirious hallucinations as based on disequilibrium of the endocrine-sympathetic system which controls emotivity. As Tinel, Santenoise and Garrelen believe, the disturbance may be one of a vagotonic preparatory phase followed by a compensatory phase of extreme sympathicotonia. On the other hand, the slight mental deficiency renders inaccurate the control of the higher psychic functions, the patient accepting easily the suggestions of his mental automatism. Finally, the poor physical condition and the endogenous intoxications may explain the presence of hallucinations which differentiate this variety from other forms of acute delirious psychosis.

FERRARO, Washington, D. C.

SOLE PATTERNS OF TWINS. R. B. MONTGOMERY, Biol. Bull. **50**:293 (April) 1926.

"In the study of twins, one frequently desires to know whether or not they are of monozygotic or dizygotic origin." In the newly born, examination of the placenta and membranes answers this problem, but in children and adults other methods are necessary. In such cases the study of the general physical appearance, mentality, palm patterns, whorls of the hair, etc., have led to no definite results, and Montgomery undertook the study of sole patterns in the hope of revealing some means of identification.

Prints were secured from eighty-seven pairs of twins by a process perfected by Mathews. The age of the youngest pair was 1 week, and of the oldest, 85 years. Most of the subjects, however, were school children. Of the eighty-seven pairs of twins, thirty-three pairs were male-male, twenty-four were female-female, and thirty were male-female. Newman thinks that monozygotic twins are likely to have identical patterns, but that unlike patterns do not disprove monozygotic origin. Wilder also has remarked on the striking similarity which sometimes occurs but declares that, while they are similar grossly, the Galton details are always different. Montgomery found thirteen sets in his series of eighty-seven pairs of twins with identical patterns, or a percentage of 14.94. In all except one pair, this similarity of pattern occurred in like sex twins. He concludes that "the presence of identical patterns on the soles of a pair of twins might point to their monozygotic origin, but as Newman states, their absence does not disprove it."

ALPERS, Philadelphia.

REMARKS ON A CASE OF CHRONIC ANTERIOR POLIOMYELITIS TRANSITORY TO AMYOTROPHIC LATERAL SCLEROSIS. A. FOIX and J. A. CHARANY, J. de neurol. et de psychiat. **26**:83 (Feb.) 1926.

The authors describe a case of chronic poliomyelitis which during life was accompanied by signs of pyramidal tract involvement (tendency to clonus and to the Babinski sign). These signs caused a suspicion of amyotrophic lateral sclerosis. The diagnosis was, however, discarded because of the unusually long course of the disease. From the pathologic point of view, the main aspects were the lesions of both anterior horns of the spinal cord. The

nerve cells were largely involved and atrophied, especially in the cervicodorsal region. Besides the lesions of the nerve cells, there was an evident degeneration of the pyramidal tract at the level of the sacral region. The involvement of the pyramidal tract was moderate and localized.

The question arises whether we are dealing here with a stationary lesion or with one that is extremely slow in evolution and the result of which becomes manifest late. The fact that the slight pyramidal signs had been present since the early stages of the disease makes the authors incline toward the first hypothesis. They emphasize this combination of lesions of typical chronic poliomyelitis with moderate localized lesions of the pyramidal tracts in order to raise the question of the transitional forms between chronic poliomyelitis and amyotrophic lateral sclerosis. According to their point of view, no barrier exists between the two diseases.

FERRARO, Washington, D. C.

THE PREVENTION AND CURE OF TETANY BY ORAL ADMINISTRATION OF STRONTIUM. W. W. SWINGLE and W. F. WENNER, Am. J. Physiol. **75**:378 (Jan.) 1926.

This investigation was conducted on a series of twenty-seven thyroparathyroidectomized dogs, carefully controlled as to diet. Immediately following operation and after the effects of the anesthetic had abated, 200 cc. of 5 per cent strontium lactate was administered to each subject by stomach tube and the same amount exhibited from then on daily. It was found that by this procedure tetany was preventable and, in instances in which it had already occurred, curable if combined with bleeding. This the authors explain as dependent on the sedative influence of strontium and the decrease it induces in intestine permeability to calcium excretion, thus permitting the maintenance of a satisfactory calcium content parenterally, on restoration by bleeding as was actually determined in this work. It was found further that after a period of about forty days of strontium medication, animals were able apparently to go on indefinitely without developing tetany in the absence of additional strontium administration. In explanation of this observation, the authors suggest the possibility of the institution of vicarious function on the part of other tissues, also the possibility that existing accessory or residual parathyroid elements may, after a transitional period, be able to assume and carry the entire normal functional load.

RAPHAEL, Ann Arbor, Mich.

THE DEPENDENCE OF SECONDARY SEX CHARACTERS ON TESTICULAR HORMONES IN LEBISTES RETICULATUS. L. J. BLACHER, Biol. Bull. **50**:374 (May) 1926.

Secondary sex characters depend in their development on hormones produced in the sex glands. After castration, these characters will disappear. While this has been shown in other animals, only recently has it been attempted in fishes. Kopeć castrated *Phoximus laevis,* males and females, and found a failure of development of the nuptial colors. The hybridologic analysis of the sex character in fishes was carried out by Aida on *Haplochitris latipes* and by Winge on *Lebistes reticulatus.* Both authors concluded that the color characters of the male depend on the genes located in the sex chromosome, especially in the Y-chromosome.

Blacher studied the effect of the testes in *Lebistes reticulatus,* using seven fishes, six of which were males. He found in the six males that the atrophy of the testis is paralleled by the disappearance of the sex colors, and concluded that the intensity, shape and development of the black, and especially the red

and yellow, pigment spots in these fishes is dependent on hormones produced in the testis. With the disappearance of the male sex coloration after castration, the individual approaches the coloration of the female.

ALPERS, Philadelphia.

THE EFFECT OF THYROIDECTOMY ON SOME UNCONDITIONED RESPONSES OF THE SHEEP AND GOAT. H. S. LIDDELL, Am. J. Physiol. **75**:579 (Feb.) 1926.

An attempt was made in this investigation to determine the effect of thyroidectomy on both conditioned and unconditioned reflexes in goats and sheep. The unconditioned response studied was the defensive reaction of the foreleg to tetanizing faradic stimulation, and the conditioned response the reaction to minor tactile stimuli of a magnitude below the unconditioned threshold. Liddell discovered that in cretinized animals the unconditioned or "inborn" reflex persists to the point of death, while the acquired pattern becomes less and less effective with intensification of the athyroid syndrome. He suggests further that the general behavior (lethargy and reactive instability) of thyroidectomized animals may possibly be dependent on diffuse cerebral inhibition resulting from the metabolic disturbance consequent to thyroid deprivation. This study, although still in a relatively preliminary phase, is stimulating and thought provoking, lending itself to interesting speculation relative to the nature of conduct, particularly pathologic conduct as incident to various neuropsychiatric syndromes such as, for example, the regression phenomena of schizophrenia.

RAPHAEL, Ann Arbor, Mich.

THE POSSIBLE RELATIONSHIP BETWEEN ACROMEGALY AND DIABETES. H. T. JOHN, Arch. Int. Med. **37**:489 (April) 1926.

The author reports two cases of acromegaly associated with diabetes and reviews other cases in the literature in which such association has occurred. In general, hypopituitarism is accompanied by increased carbohydrate tolerance and hyperpituitarism by decreased tolerance, although exceptions to this rule occur. The onset of acromegaly tends to precede the onset of the diabetes by many years. Diabetes in these cases is treated in the usual way, and response to treatment presents no differences from the ordinary case; if treatment is not properly instituted these patients are similarly subject to acidosis and coma. The cause of the diabetes appears to be hyposecretion of the posterior part of the pituitary gland which in some way tends to decrease the insulinic secretion, although hyperpituitarism and decreased insulinic secretion may not result from the same unknown factor.

VONDERAHE, Cincinnati.

RESULTS WITH TRYPARSAMIDE IN THE TREATMENT OF NEUROSYPHILIS. LOUIS BERG, Am. J. Syph. **10**:261, 1926.

After reviewing briefly the chief contributions to tryparsamide therapy by previous workers, the writer discusses the treatment of neurosyphilis by this drug among patients at the Manhattan State Hospital. His patients were divided into two groups: one group treated by simple intravenous administration of tryparsamide, and the other with this drug combined with hypertonic sodium chloride solution and drainage. He can detect no certain difference in the results of treatment with the two methods. He is convinced, however, that approximately 40 per cent of unselected hospital patients (excepting only

those found undesirable on ophthalmoscopic examination) show clinical improvement. This improvement is by no means always paralleled by serologic observations.

ANDERSON, Philadelphia.

THE RÔLE OF THE NUCLEUS IN THE CELL FUNCTIONS OF AMEBAS. E. B. BECKER, Biol. Bull. **50**:382 (May) 1926.

Becker found that enucleated amebas of the species *Amoeba dubia* show the following properties: (1) within a few minutes after enucleation streaming ceases, and the ameba becomes a wrinkled sphere; (2) in a few hours the streaming is resumed; (3) this streaming is usually of the limax type, though it may approximate the normal; (4) enucleated amebas may attach themselves to the substriatum; (5) enucleated amebas are irritable, but do not show the same response to stimuli which nucleated amebas show; (6) food organisms which enter the body of an enucleated ameba are killed and digested in an apparently normal manner; (7) enucleated amebas may become divided by antagonistic streaming currents within.

ALPERS, Philadelphia.

THE EFFECT OF BLEEDING ON THE SERUM CALCIUM OF THYROPARATHYROIDECTOMIZED DOGS. W. W. SWINGLE and W. F. WENNER, Am. J. Physiol. **75**:372 (Jan.) 1926.

Studies made by these workers on a series of seven thyroparathyroidectomized dogs demonstrated that bleeding (from 30 to 110 cc.) appears to induce speedy relief of tetanic symptoms. This relief, which is temporary (from ten to twelve hours), apparently is not due to the dilution of blood toxin, as had been previously supposed by some, but rather is brought about by a striking elevation in the blood calcium content. Thus for the entire series, the average calcium content rise, from bleeding, was 20.8 per cent. Return of symptoms was coincident with the return of the blood calcium to the original low level.

RAPHAEL, Ann Arbor, Mich.

THE TREATMENT OF SYPHILIS OF THE CENTRAL NERVOUS SYSTEM. FREDERIC J. FARNELL, Am. J. Syph. **10**:236, 1926.

The author outlines his procedure of extradural injections of arsphenamized serum. This method he regards as hopeful because of the presence of a dialyzable membrane and the blood supply; he has used it in a variety of cases of syphilis of the spinal cord. He recommends the intravenous use of hypertonic iodide solution as a means of avoiding gastric irritation, and speaks most favorably of its value in even advanced tabetic scleroses. An interesting and important feature of his discussion is his emphasis on the treatment of the individual and his disease.

ANDERSON, Philadelphia.

Society Transactions

PHILADELPHIA PSYCHIATRIC SOCIETY

Regular Meeting, May 14, 1926

EDWARD A. STRECKER, M.D., *President, in the Chair*

GENERAL PARALYSIS: A CLINICOPATHOLOGIC STUDY WITH ESPECIAL REFERENCE TO THE SIGNIFICANCE OF THE SO-CALLED "TYPICAL" LABORATORY OBSERVATIONS. DR. N. W. WINKELMAN and DR. GEORGE WILSON.

A series of sixty-seven cases of general paralysis studied clinically, serologically and pathologically was presented as follows: (1) thirty-nine in which the diagnosis of general paralysis was made on the basis of the history and the physical and laboratory examinations, and which was proved by microscopic studies; (2) six cases with the so-called typical results of laboratory observations, proved by examination of the brains to be general paralysis, but diagnosed incorrectly; (3) twelve cases, diagnosed general paralysis, some of which showed general paralysis at the laboratory examination but in which this disease was not found; (4) eleven cases in which the diagnosis of general paralysis was considered and in which conditions observed in the laboratory were suggestive, but the cases were neither so diagnosed nor did they show this disease on microscopic study.

The six cases in group 2 had the following clinical diagnoses: right cerebral hemorrhage and tabes; amebic abscess of the liver and tabes; syphilitic meningitis; tabes; syphilitic meningomyelitis; dementia praecox.

The twelve cases of group 3, incorrectly diagnosed as general paralysis, had the following microscopic diagnoses: cerebral syphilis; arteriosclerosis of the small vessels; arteriosclerosis with multiple areas of softening; cervical hypertrophic pachymeningitis; meningovascular syphilis with areas of softening; gumma of the left frontal and softening of the right frontal lobes; arteriosclerosis with areas of softening; arteriosclerosis with thrombosis of the superior longitudinal sinus; thrombosis of the middle cerebral artery with softening; meningovascular syphilis; glioma and arteriosclerosis.

The conclusions drawn from this study were: 1. An untreated case of general paralysis will show blood and spinal fluid evidence of syphilis; these tests will usually be markedly positive including pleocytosis, an increased amount of globulin and the paralytic gold curve. 2. A diagnosis of general paralysis without strong confirmatory laboratory evidences in an untreated case is practically always wrong. 3. A certain limited number of cases showing mental symptoms and evidences in the blood and spinal fluid of a paralytic nature, on microscopic study are found not to be cases of general paralysis. 4. A brain which shows a gross organic condition such as hemorrhage, thrombosis or gumma practically never shows microscopic evidences of general paralysis. 5. Strong laboratory evidences of syphilis without mental symptoms are not enough to make the diagnosis of incipient general paralysis (paresis sine paresi).

DISCUSSION

DR. JOSEPH V. KLAUDER: The essayists should be congratulated for this study, which must have entailed considerable work and is of great value. I

know of no similar study reported in the literature although much has been written correlating the spinal fluid observations with the clinical picture. The analysis speaks in favor of the significance to be attached to the characteristic laboratory observations. What constitutes the typical laboratory picture? The blood Wassermann reaction appears to be 4 plus in almost 100 per cent of cases. There are only two other stages of syphilis in which this holds true: active congenital syphilis, and the secondary eruptive stage of the disease. In the spinal fluid, it is the composite picture, the results of all the tests rather than of any one. Less than 100 lymphocytes per cubic centimeter appears to be characteristic. Plasma cells are likely to be present. The globulin test is uniformly positive. The fluid characteristically yields a 4 plus Wassermann reaction in dilutions varying from 0.1 to 1 cc. The last feature should be stressed; this and the gold curve are the most distinctive observations. The paralytic gold curve, as stated by the essayist, may be present in neurosyphilis other than general paralysis and also in other neurologic conditions.

In considering the significance of the laboratory examinations, it is appropriate to discuss what Southard and Solomon called paresis sine paresi, in which typical laboratory observations exist in the absence of other symptoms that would justify a diagnosis of general paralysis. I saw a patient in the late secondary stage of syphilis who could be placed in this category. The status of such patients cannot be stated definitely. Spinal fluid abnormalities may precede clinical evidence of neurosyphilis for a number of years, and it may be that an abnormal spinal fluid, typical of general paralysis, represents the "preparetic" stage of the disease. However, it will require much study before the significance of the paralytic gold curve in this class of case can be interpreted. One can neither deny nor affirm that these cases are "preparetic." Contrasted to this type are the cases mentioned by Steiner, in which both the blood and spinal fluid in latent syphilis are negative, but rather suddenly become positive with the simultaneous appearance of symptoms of general paralysis.

Dr. F. X. Dercum: The paper is both interesting and instructive. Without stating the facts with so many words, the authors have emphasized the great difference between the paralytic or parenchymatous form of cerebral syphilis and the gummatous form. In considering the tables presented there will, I think, be little difficulty in making distinction between the cases of the exudative form of nervous syphilis and those of true general paralysis. The difference between the two forms of syphilis is, I believe, fundamental. Indeed, I would not be surprised if the future did not demonstrate that there is a difference between the spirochetes which give rise to the two different diseases. A very suggestive experience of mine is the statement from certainly half a dozen of my patients that they have had syphilis twice; it has occurred to me that possibly they had been infecteed at one time with the exudative and at another with the parenchymatous form. It is noteworthy, also, that, in the pathologic reports presented, in no instance have there been in the same case lesions of both the parenchymatous and the gummatous or exudative form of syphilis.

Dr. J. Hendrie Lloyd: Two conditions ought to be thought of in this connection. First is what we used to call syphilitic dementia. This is a syphilitic dementia that is not general paralysis and is recoverable. Many years ago, a man under my care had many symptoms suggestive of the early stage of general paralysis (it was long before the days of the Wassermann test), and gave a clear history of a primary sore. He was given treatment with iodides and mercury and made an astonishing recovery. He lived thirty years afterward, and was in active business. The second condition is called

pseudoparesis, which can usually be ruled out nowadays with laboratory tests. It occurs in alcohol and drug addiction. The patients pass into a condition suggestive of general paralysis, with expansive delusions, tremor of the face, etc. As a rule, they do not have Argyll Robertson pupils. I recollect a physician, a friend of mine, who for several years had been taking drugs and alcohol; I sent him to the Pennsylvania Hospital for the Insane. He had tremor of the face and hands, and mild expansive delusions; I think the pupils also were rather sluggish. In a few weeks he made a practically complete recovery.

COLERIDGE AND HIS OPIUM HABIT. DR. J. HENDRIE LLOYD.

This paper, showing the evil effects of the excessive use of the drug by the English poet, was not discussed.

A. CURRENT CONCEPTIONS OF BODILY STRUCTURE IN RELATION TO TEMPERAMENT AND CHARACTER. DR. G. E. PARTRIDGE and DR. C. B. FARR.

B. ANTROPOLOGICAL METHODS IN PSYCHIATRY. DR. C. B. FARR and DR. H. MARSHALL.

C. ANALYSIS OF OLDER ANTHROPOLOGICAL RECORDS. DR. S. M. BARON and DR. C. B. FARR.

D. FUNCTIONAL CARDIAC TESTS IN THE DIAGNOSIS OF THE PSYCHOSES. DR. H. MARSHALL.

Dr. Farr summarized *A*, *B* and *C*. He first defined and discussed the terms used in studies of constitution, such as: constitution, "condition," personality, temperament, character, physique, habitus, etc. The term "constitution" was defined to include only those peculiarities of physique, temperament and character that are inherited; "personality" to include both hereditary and acquired characteristics. As used by psychologists, "personality" does not include physical characteristics. The various terms used to designate habitus were then discussed, with particular reference to those adopted by Kretschmer; that is, the "asthenic" or slender type, the "pyknic" or thick-set type, and a third type, for which Kretschmer himself is responsible, which he designates as the "athletic" type. The peculiar physical types that are clearly due to endocrine factors he classifies together as "dysplastics." Clear-cut types are rare and mixed forms common, as would be expected from the laws of heredity. Kretschmer worked with an almost pure racial type; in Farr's material the influence of race was confusing. The racial types — Nordic, Alpine and Mediterranean — were mentioned, and the various older classifications of habitus compared. An attempt was made to make correlations between structure, physiologic function and behavior reactions. Under the first heading were classified general physical examination, roentgen-ray observations, anthropometry, etc.; under the second, "functional" and psychologic tests, and under the third (behavior), intelligence, drives, attitudes, adjustments (Hoch and Amsden). Anthropometric measurements and indexes, as determined in the twenty-five men suffering from dementia praecox and manic-depressive psychoses, were exhibited by means of charts. Typical "asthenic" types occurring in dementia praecox, and equally typical "pyknic" types occurring in manic-depressive psychoses, were shown. In this small series the evidences seemed to correspond with Kretschmer's views, particularly with respect to the frequency of the typical asthenic habitus in schizothymia, and its comparative

rarity in cyclothymia. In these patients some seventy-five measurements and indexes were used. The "constitutional index" of Pignet seemed to be the most significant single index. The most useful information was obtained by comparison of graphic charts (constructed from measurements) with direct observation of the patient himself. One hundred and twenty older records in women, in whom less elaborate measurements had been made and in whom the opportunity had passed for direct comparison, gave disappointing results. It appeared that the sex characters in the woman, as pointed out by Kretschmer, tended to obscure the inherited physical type. A series of anthropometric measurements, sufficiently detailed, are now being carried out in women suffering from functional psychoses.

Following Dr. Farr, Dr. Marshall (*D*) discussed the functional cardiac tests of Trentzch. His series was not large, but confirmed previous observations, indicating a normal cardiac response in the subjects of dementia praecox. In a series of boys suffering from behavior disorders, the result of epidemic encephalitis, the functional cardiac tests gave normal results.

Dr. Partridge emphasized the need of a comprehensive term to include physical and psychic "make-up" — a practical concept which would not be determined entirely by the desire to keep separate constitutional, i. e., inherited as contrasted with acquired traits, but which would represent a developmental or genetic point of view. He spoke also of the need of a working hypothesis, or plan of work, which could help to avoid indiscriminate gathering of data that we might not be able to bring into practical synthetic form.

DISCUSSION

Dr. F. X. Dercum: The subject is too large to take up in a systematic manner. We have all been impressed by the morphologic peculiarities presented by many of the insane. No doubt, in a measure, these are related to concomitant endocrine anomalies. Extensive experience in necropsies in cases of dementia praecox and, to a less extent, in cases of manic-depressive psychoses, lead to an inference justified, I believe, by the observations: that these cases present on the whole a picture of morphologic subnormality. In the many necropsies I made in Norristown, years ago, I was not infrequently impressed by the small size of the heart and the small size of the larger blood vessels and also by occasional visceral anomalies, often gross in character: unusual fissuration of the liver, lobulations of the kidneys and once, as I recall, a true horseshoe kidney; these experiences were not the same as in necropsies made at other hospitals.

In former years we did not give sufficient attention — and perhaps we do not yet — to the endocrine glands in our necropsies. Often, I am sure, anomalies such as are presented by a persistent thymus are overlooked or are not regarded as significant. I have always regarded the rather frequent finding of a more or less enlarged thyroid as directly related to a thymic persistence. Thymic persistence itself must be regarded as an index of morphologic subnormality, failure of development of the organism as a whole. It is hardly necessary to emphasize the general physical symptoms which are presented by so many of the insane — especially those in the dementia praecox group — such as vascular asthenia, lividity and other cardiovascular anomalies which refer to suprarenal inadequacy. It seems hardly necessary, also, to touch on the rôle which is at times unquestionably played by the pituitary gland in these cases.

Just what is to be accomplished by the elaborate measurements that have been exhibited I do not know, unless it is by a comparison with normal

persons; here again the enormous spontaneous variation among normal persons will greatly lessen the value of the results or at least make their interpretation difficult. Normal human beings vary enormously in their individual morphology, and to attempt to fix an absolute standard of development seems futile. Indeed, variability is a normal characteristic of the human race, a characteristic that is expressive of its marvelous and inherent plasticity and which has made possible its adaptation to the most varied conditions of its environment, geographic, climatic, nutritional, etc., which have demanded in times past and still demand increasing changes in adaptation.

Dr. Edward A. Strecker: A number of years ago, Frederick Mott reported testicular atrophy in dementia praecox. The only comparable pathologic change he could find was in senile dementia.

Dr. H. L. Stick: About fifteen years ago, I attempted to make such measurements with reference to different types of mental disease. I examined and measured about 2,000 patients, but never tabulated the observations. In making these measurements, we must start with a norm in order to come to a definite conclusion. Possibly the old Greeks made measurements and then started from that norm as we are now attempting to do.

Dr. Strecker: This method of scientific study, which is only beginning in psychiatry, has been going on for a great many years in medicine, so that types of persons who are likely to develop pulmonary tuberculosis or gall-bladder disease, and so on, are being defined. If we can predict approximately what a certain person will develop, there may be some chance of preventing it. That is a practical consideration.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, May 28, 1926

W. G. Spiller, M.D., *President, in the Chair*

A Case of Local Sweating Confined to the Partial Distribution of the Ulnar Nerve. Dr. R. W. Waggoner.

A girl, aged 15, came to the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases complaining of recurrent attacks of sweating affecting, as will be shown, only part of the ulnar and internal cutaneous distributions of the right arm. The family history was essentially unimportant although the mother states that she herself blushed easily and perspired freely when young. The patient was born normally at term; it is stated, however, that one hand was pressed so tightly against the cheek that the imprint lasted for a day or two. The mother is not clear as to whether this was the right or the left hand. The developmental history was normal. The child walked and talked at 13 months. When about 18 months of age, she was caught under the side of the crib, but there was no history of injury at that time. At 5 years, she had a rash which was diagnosed German measles, but it lasted for eight weeks. At this time, she also had a severe cold with considerable nasal discharge. At 7 years, she had an abscess under the right angle of the jaw, and at 8 years she had a similar condition on the left side. During

her eighth year, she had scarlet fever and diphtheria. The mother stated that the girl was easily excited and upset and rather irritable.

At the age of 3, the patient fell on the floor and bumped her right hand against a hot stove door. The skin of the hand over an area about the size of a silver dollar, just proximal to the fourth metacarpophalangeal joint was badly burned. Shortly after this wound had healed, the mother noticed that at irregular intervals there would be an excess of moisture over an area about the size of a quarter just below the styloid process of the ulna. At first the sweating occurred only occasionally, but when the patient started to go to school it was noted that the sweating became much more frequent and involved an increasingly larger area on the arm. Since that time the sweating had been present only during the school months, from September to May. The sweating was most marked when she was excited or nervous, or when she attempted to write or to work with that hand. It was sufficiently profuse to soak the paper on which she was writing. In winter, when she was outdoors, it appeared at times as if steam were coming from the areas on her arm, and she had noted bits of ice over the sweating area. The sweating usually lasted for about one hour. The area involved often blushed when she was excited or nervous without actually sweating.[1] While sweating, the area would become white and cold, but about fifteen or twenty minutes after the sweating had stopped it would regain its normal appearance.

Physical examination gave practically negative results. The girl was large and over-developed, having the appearance and actions of a girl of 17 or 18. At the time of the first examinations, there was rather marked acne on the face. The thyroid gland was slightly enlarged, and the pulse rate was somewhat increased. There was no tremor of the hands on extension. No symptoms of cranial nerve involvement were found. The deep tendon reflexes in the upper extremities were normal, but those of the lower extremities were somewhat increased, equally on the two sides. The skin reflexes were normal. At the time of the first examination, the sweating area was noted as extending from the metacarpophalangeal joint of the little finger to about 2½ inches (6.2 cm.) above the styloid process of the ulna. The area was ovoid but rather irregular in outline and was about 2 inches (5 cm.) wide in its widest part; the margins were sharply defined. All forms of sensation over the area involved were normal. The patient entered the hospital, April 13, 1926, and remained for about a month. During that time, she gained 14½ pounds (6.5 Kg.). None of our examinations revealed anything of note; the urinalysis, the blood count, the blood chemical determinations and the blood Wassermann test gave negative results; the basal metabolic rate was plus 6. Roentgenograms of the skull revealed a normal sella, with no evidence of intracranial disease; roentgen-ray examination of the right hand, right arm, right shoulder joint, cervical and thoracic vertebrae and of the upper thoracic region showed no disease or abnormal condition of the bones. Electrical examination of the right arm gave normal results. Intramuscular injections of 1/20 grain (0.003 Gm.) of pilocarpine resulted in no excessive sweating either local or general, and practically no change in blood pressure or pulse rate. Intramuscular injection of 1/10 grain (0.006 Gm.), however, caused a slight rise of blood pressure with a rather marked drop of the pulse rate from 110 to 80 a minute. At the end of ten minutes the area of sweating began to feel warm, subjectively; at the end of twenty minutes, the patient noticed a general warmth and at the end of twenty-eight minutes the area on the arm began to sweat. This sweating lasted for about forty-seven minutes. There was no general sweating. At the end of

one hour, she felt normal and showed no visible evidence of vasomotor disturbance.

The next day the patient was given 1 cc. of 1:1,000 solution of epinephrine hydrochloride intramuscularly just as the arm was beginning to sweat. At this time the arm was moist; at the end of ten minutes the sweating had stopped and at the end of twenty minutes the arm was dry. No subjective change of sensation was noted. The blood pressure and pulse rate showed a distinct rise at the end of thirty minutes but returned to normal at the end of one hour.

During the first three weeks at the hospital, the patient complained that the hand was sweating more frequently than it had at home. On May 8, there was no sweating, and she remained free from the symptom until May 15, when there was one attack which was more marked than any previous one since her admission to the hospital. She has had no sweating of the arm since that time.

The cause of this condition is obscure. A number of theories might be offered, but I doubt that any could be proved. In a review of the literature, I have been unable to find a similar case. A number of cases of unilateral sweating followed hemiplegia; in a case of local sweating of the face necropsy showed definite organic disease of the corresponding sympathetic ganglion. There have been cases of sweating of one extremity in lesions of the cord and also cases in which the distribution of a single nerve is involved following injury to that nerve.

Raymond, in describing local sweating of the face, states that it may result from one of four factors: (1) material alteration of the cerebrospinal nervous system such as general paralysis, hemiplegia, cerebral congestion or epilepsy; (2) lesions of the cervical sympathetic nerve of the affected side, such as may be caused by aneurysm, after ligature of the carotid, hypertrophy of the thyroid, exophthalmic goiter or sinus thrombosis: (3) lesions of the peripheral nerves by wounds; (4) reflex, as the result of a disorder of some other part, or from psychic disorders. Weir Mitchell reported a case of excessive sweating of one arm following gunshot injury of the sympathetic nerve on that side and the same symptom after reflex paralysis of the affected arm. Crocker inferred that local sweating is due to a disease or injury which directly or indirectly interferes with the function of the sympathetic nerves on the side affected. Hare stated that local sweatings occur almost solely in nervous disease, and that they depend on perverted vasomotor influences sent to the glands and their supplying vessels in the areas involved. Sahli said that similar disturbances of sweat secretion may occur in syringomyelia. Burns stated that sweating is often increased after degeneration of the sympathetic nerve supply; he believed that it is possible to have changes due to defective peripheral circulation.

De Bec Turtle reported a case of suppuration of the parotid gland which was followed by a salivary fistula; this, however, healed. After that, whenever the patient ate, sweating would occur in the distribution of the auriculotemporal nerve. Parkes Weber thought this was due to sympathetic nerve fibers caught in scar tissue, which were irritated during eating. Rhein reported a case of unilateral sweating of the face in which he inclined to the diagnosis of hysteria. Paudi concluded that sweating is controlled by a cortical center, to which are carried impulses from the periphery through the anterior roots and the motor fibers of the sympathetic system; that abnormal sweating can

be caused by either irritation or interruption of the transmitting fibers, and that the cause of sweating when there is obstruction in the pathway is due either to degeneration of the sweat glands above the palsied parts or to psychomotor influences.

Dr. Mills suggested to me the possibility of a weakness or lesion in a certain distribution of the sympathetic system involving this area, or of a possible arrest in development of a certain portion of the sympathetic system.

In this case, we have examined the patient in vain for some organic cause of the condition. There is no evidence of any disturbance of the sympathetic system. The symptoms are much more marked when there is some psychic disturbance. It seems logical to conclude, therefore, that the sweating may be due to some weakness or defect of the sympathetic system supplying this area, in which the application of the stimulus of an emotional stress results in the excessive sweating.

DISCUSSION

Dr. Charles K. Mills: I have had under observation in the course of years a number of cases of unilateral or localized sweating, one or two of which I have reported. This phenomenon may be present, I believe, in cases of lesions variously situated in the spinal cord, in the sympathetic ganglia and in the peripheral nerve itself. In this case the probabilities are that the original peripheral nerve lesion had impaired the nerve supply. Localized sweating may occur from lesions anywhere along the spinal cord in its thoracic or sacral portion concerned with vasomotor and secretory functions. Of course, there is a psychic factor in this case. I do not believe, however, that such cases are often purely hysterical.

Postencephalitic Ataxia. Dr. A. M. Ornsteen.

Case 1.—A white man, aged 45, became acutely ill with epidemic encephalitis in February, 1925. The onset was interesting because the man awoke feeling as well as usual; while washing, he experienced a most peculiar sensation as though his abdominal viscera were suddenly pulled out of him; he then had severe vertigo and fell to the floor. He became confused and delirious that afternoon and subsequently went through a severe course of the acute form of the disease lasting about six weeks. On leaving bed, he was unable to stand or walk because of vertigo and staggering; this condition has persisted to the present time, but is greatly ameliorated. He vomited in the mornings for several months; this symptom has disappeared entirely.

The clinical picture of the postencephalitic state, when I saw him, was typically of a cerebellar character. The gait was unsteady and wide-based, with inability to stand with the feet approximated. In getting up from a sitting position preparatory to walking, the patient would lurch quickly to the left to prevent falling. When the trunk was bent backward, the knees were not flexed as much as in the normal associated act. He was unable to walk around a central object without either diverging from it when going clockwise, or running into it when going counter-clockwise. The patient was able to lie on his back with the feet elevated, and the hips and knees flexed for one hour and fifty-five minutes as compared to three minutes in a normal man placed beside him (cerebellar catalepsy of Babinski). The deep reflexes were normal; there was no change in muscle tonicity; there were slight lateral nystagmoid movements of the eyes in both directions. The pupils, eyegrounds and cranial nerves were normal. The results of serologic examination of the blood and spinal

fluid were negative. The vestibular observations suggested brain stem lesions. There were no tremors and no parkinsonian facies or attitude.

The man is gradually improving; the cataleptic phenomena have disappeared in the last two months; he is less dizzy, and the gait has improved.

Case 2.—The onset in this man, aged about 48, also occurred in the morning soon after arising, in the form of numbness in the left foot which gradually extended up the left side of the body, limbs and face within several hours. The man also became excited, delirious and confused the same afternoon, vomiting in a projectile manner. He subsequently presented an acute febrile encephalitic picture with cranial nerve palsies (left seventh and sixth and right third nerves, and later a suggestion of bulbar involvement because of moderate hoarseness and dysphagia). Associated with this was left hemiparesis of the pyramidal type and left hemianesthesia for both superficial and deep sensibility. After eight weeks the patient reached the state he now presents.

The postencephalitic picture when I last saw him was a left hemianesthesia with ataxia of severe grade interfering greatly with locomotion. There was complete loss of position and passive motion recognition in the upper limb up to and including the elbow, but most marked in the hand and wrist; the condition was also present in the left lower limb. Astereognosis in the left hand was complete. He was unable to put his left forefinger to the nose because of the extreme ataxia. The motor impairment had receded in degree, although there were hypertonicity and spontaneous choreo-athetoid movements in the hand and less in the toes of the affected side. The right side was unaffected. There was still evidence of the previous extra-ocular palsies, but the facial innervation was good. There were still heminumbness and slight impairment of the touch, pain and temperature senses in the distal portions of the left limbs particularly.

The first patient had an ataxia due, probably, to involvement of the fibers of the cerebellorubrospinal pathways in the brain stem but, because of the preponderant affection of the trunk as compared to the ataxia of the limbs, the cerebellum itself might be the seat of the degenerative process.

In the second patient, the lesion responsible for the hemiataxia and allied symptoms was in the right thalamostriatal area.

A Case of Cortical Monoplegia. Dr. F. N. Anderson.

A painter, aged 69, was admitted to the outpatient clinic of the Orthopaedic Hospital on March 5, 1926. His chief complaint was "helpless left hand." In May, 1925, he fell in a drug store shortly after drinking some iced beer. He said that he did not lose consciousness, and that he had no difficulty in walking or in talking. The left arm, however, was paralyzed. He was taken home, and on the following day was admitted to the hospital. At this time he thinks he may not have been mentally clear, as some things were later told him which he did not remember. He was in the hospital three weeks.

He has improved as far as the upper arm is concerned, but his hand is almost as helpless as after the onset.

The previous history was essentially unimportant, although there was a vague history of a penile lesion forty years previously.

Examination.—The patient was tall and emaciated. Snags of teeth, opacities over the right pupil, poor chest expansion, generalized abdominal tenderness and bilateral hernia were noted. The left upper extremity was held in a rather strained position, with the fingers extending at the interphalangeal joints and showing movements of choreo-athetotic character. The seventh cranial nerves

seemed weak, but equally so. The deep reflexes were increased, more on the left. No abdominal or cremasteric reflexes were obtained, but the plantar reflexes were normal. Dynamometer readings were: right, 80; left, 20. The arms were of normal strength.

Sensory examination was uncertain, but perception seemed more acute for light touch and pinprick over the left forearm and hand.

Clinical Course.—In a sensory test on April 16, no differences could be made out. May 14, the left hand was found to be impaired as follows: (1) two point discrimination: right fingers at 1 cm., left at 2 cm.; (2) light touch: greater on the right, but chiefly diminished over the left hand; (3) pinprick: less over the left hand; (4) vibration: no difference; (5) position: uncertain; fingers stiff.

On May 28, passive movement showed definite and marked impairment in the fingers of the left hand, constituting the sharpest differential point encountered. Partial astereognosis was detected, as the patient could not recognize small objects in the left hand. When tested for temperature sense, he uniformly said cold was colder and warm warmer on the right than on the left, but he always called the correct temperatures on the left.

In crossing from side to side in a contiguous point series to establish a line of demarcation, I have been unsuccessful. It seemed certain that there was a distinct sensory impairment of the distal portions of the left upper extremity, becoming less pronounced as one proceeded proximally.

SPECIMENS SHOWING LOCALIZED CORTICAL LESIONS: (1) LIMITED TO BROCA'S AREA, (2) LIMITED TO THE MOTOR FACE AREA. DR. R. B. RICHARDSON.

The author presented two gross brain specimens, because they showed well marked and sharply defined cortical areas of softening, with equally definite clinical symptoms. In the one case, there was complete loss of the power of speech, then redevelopment of this power after an absence of months, most progress occurring just previous to the time of death. In the other case, the only clinical symptoms were the concomitant onset of weakness of the right side of the face and stammering.

CASE 1.—N. P., an ex-medical student, white, aged 52, was admitted to the Philadelphia General Hospital, March 9, 1926, and died May 2, 1926, following an operation performed to relieve a condition clinically diagnosed as volvulus. Ten days previous to admission to the hospital, he lost the power of speech. He was confused, unable to utter a word and had definite weakness in both right extremities. If facial weakness was present, it was not noticed. When he was admitted, the weakness in the extremities had disappeared, but the power of speech was completely absent. The man had difficulty in understanding spoken words and in obeying complicated commands; but he was able to write a few words, and expressed himself by this means. On writing from dictation he omitted and misspelled words. By April, he was rapidly regaining his power of speech, when he was suddenly seized with a condition that on necropsy was revealed to be a necrosis of the sigmoidal portion of the bowel. Operation failed to relieve this condition.

Examination on the date of admission revealed a poorly nourished man, unable to speak, but able to make expression as indicated above. The heart sounds were distant and of poor quality; the blood pressure was 120 systolic, 60 diastolic, with vessels moderately sclerosed. The pupils were contracted, and reaction to light and in accommodation was normal. The conjunctivae on the right presented a questionable diminution of sensation. The tongue deviated

to the right; the deep reflexes were equal and active, and there was no Babinski reflex. The sensation of the entire right side of the body seemed to be impaired, but this information was considered unreliable because of the aphasia. The motor system was normal. The laboratory examination revealed nothing of importance.

Pathology.—The cause of death has been referred to. The neurologic interest lies in the old area of softening, with cavitation due to absorption, which involved the lower portion of the left precentral convolution and the adjoining opercular and triangular portions of the inferior frontal. The softening extended inward and backward to the depth of the insula, and included the claustrum and external capsule. It was sharply limited.

Comment.—It is extraordinary that this man should have regained his power of speech to the extent that he did.

Case 2.—A white laborer, aged 70, was admitted to the Philadelphia General Hospital, Feb. 8, 1924. Death occurred on February 23, from exhaustion, the result of conditions associated with advanced age, general arteriosclerosis, cardiovascular renal lesions and gangrene of the left lower extremity.

History.—Fifteen years before admission, the man was suddenly taken with paralysis of the right side of the face and stammering. He was not unconscious. The stammering had remained constant, but the facial condition had improved. On admission, gangrene of the left lower extremity was the outstanding physical condition.

Examination.—The patient was well oriented and memory was fair, but cerebration was retarded. There was weakness of the right side of the face and ptosis of the right upper eyelid. The motor system presented slight weakness and incoordination in the lower extremities. The reflexes were prompt and equal, and sensation was normal. Laboratory examination showed: a trace of albumin in the urine; blood chemical determinations, normal; spinal fluid: cells, 0; faint trace of globulin. The Wassermann reaction was negative.

Pathology.—The observation of interest lies in the cavitation, the result of previous softening and absorption, present in the left lower motor area, which extended to the triangular area of the inferior frontal convolution. The base of the lesion lay on the sylvian fissure; it measured 4.5 by 2.5 by 3.5 cm. and extended to the full depth of the insula. The lesion was sharply defined.

Comment.—The feature of interest lies in the association with this lesion of only facial weakness and stammering. The case has already been discussed by Dr. Winkelman and Dr. Wilson in their paper on monoplegia, but is again brought to your attention in association with case 1.

DISCUSSION

Dr. C. A. Patten: Last month I presented here a case of cerebral vascular lesion with symptoms which made localization a problem. After considerable discussion as to whether it was cortical or subcortical, it seemed to be generally agreed that it probably began subcortically and extended to the cortex. It is my impression that the gross pathologic change in these cases demonstrates something more than a pure cortical involvement, and it is probable that nearly all cases show an extension of the lesion into the subcortical tissues.

The case presented by Dr. Anderson is interesting because he shows some things that cannot be explained on the basis of a pure cortical lesion. The movements of the arm and hand are, as pointed out by Dr. Weisenburg, distinctly choreo-athetoid. My conception of the pathologic change in the case of abnormal movements is that there is a lesion of the basal ganglia or related

structures. If, however, we incline to Dr. S. Kinnier Wilson's recently published views, some of these movements may be of corticospinal origin.

Dr. Charles Potts: It may be of some interest in reference to Dr. Richardson's last case to know that the patient had recovered his power of speech to a large extent. He worked in the kitchen for some time and could speak well enough to conduct a conversation. Of course, he had trouble with some words.

In reference to Dr. Anderson's case, I understood him to say that the man had impairment of tactile, pain and temperature sensation. My impression is that in pure cortical lesions there is no impairment of these forms of sensation. If I am wrong, I will be glad to know it.

Dr. F. N. Anderson: Light touch and pain sensation were definitely impaired, but not always with absolute certainty, and not to the extent of the appreciation of passive movement.

Dr. W. G. Spiller: The brain with a lesion of Broca's area was from a patient who entered the hospital in my service. He evidently had been an intelligent man and had had three years' training at a medical school. When he first came under my care, he could utter only a few words; he could not form sentences, and could write only in the manner in which he could speak. I saw him shortly before he died and was astonished at his improvement. He was able to speak in sentences slowly. The recovery of speech in a person with so much destruction of Broca's area is important. I cannot say whether he was left-handed or right-handed.

Dr. Charles K. Mills: This case showed the real part played by Broca's convolution in the symptomatology of aphasia.

Hyperreflexia of the Lower Limbs After Exercise. Dr. W. G. Spiller.

The effect of exercise on reflex activity was considered, but especial attention was paid to the exaggeration of the tendon reflexes of the lower limbs caused by slight exercise, as by walking across a room or climbing a short flight of stairs, and the possibility of this phenomenon as a sign of early degeneration of the pyramidal tracts was considered. A case was described in which the tendon reflexes in the lower limbs, when examined while the patient was in bed after a night's rest, were much less intense than they were after he was permitted to rise from his bed and walk across the floor.

The paper will appear in full in *The Journal of the American Medical Association.*

DISCUSSION

Dr. Walter Freeman: A possible explanation of hyperreflexia is to be found in the function of muscles. Each contraction of muscle generates a certain amount of waste products, chief among them being carbon dioxide, lactic acid and water, which act as a stimulus for further contractions. This is true in the heart, where waste products of one contraction are supposed to be the stimulus for the next. If, by these few contractions, a sufficient quantity of waste products could be built up in muscles, we would expect rather a condition of increased excitement of that muscle to further stimuli.

Dr. Alfred Gordon: In regard to hyperreflexia, it has been observed that in tests for the Babinski reflex, when the lower extremities are exposed to cold the plantar reflex is in flexion, but it is in extension when heat is applied. Exercise, physiologically speaking, means a larger blood supply. In both instances a greater arterial circulation may act as an irritant or stimulant

to the motor pathway, thus producing hyperreflexia. Of course, since this is observed only in a certain group of cases, it is to be presumed that in them there is probably a special state or predisposition of the nerve tracts to an exaggerated reaction.

DR. ROSS THOMPSON: Would the following be considered an instance of hyperreflexia of the lower limbs following exercise — namely, the tendency to tremor of the leg muscles following running for a train or mountain climbing? The tremor takes the form of a foot clonus when, in sitting, the ball of the foot rests on the ground with the heel raised. It disappears on rest. The reaction is similar to that of the chattering of teeth in children exposed to slight cold, except that the stimulus is thermal rather than motor.

A young woman, a stenographer, who was in the habit of sitting in a cramped position on the edge of her chair while working, one leg crossed over the other with the weight of the body supported on the ball of one foot, experienced a foot clonus, probably from fatigue. There was no difficulty in repeating and prolonging the duration of the clonus. It finally became established to such an extent that it formed the basis, in my opinion, of an hysterical shaking tremor that continued a long time.

DR. W. G. SPILLER: I think the ankle clonus described as coming on after severe exercise should be considered hyperreflexia. It is well known that ankle clonus can be produced by resting the weight of the lower limb on the ball of the foot and intentionally beginning the movement of clonus, and one may acquire considerable ability in producing this phenomenon, but there is danger that after repeated practice he may lose control over the clonus produced in this manner.

News and Comment

RESEARCH IN HUMAN BEHAVIOR

Organization of the research work in human behavior made possible by public subscription for a period of five years to the State of Illinois Institute for Juvenile Research has now been completed, and the following staff has been appointed: Herman M. Adler, M.D., Chicago, director; Horace Gray, M.D., Boston, endocrinologist; Gustav A. Jaederholm, Ph.D., Gothenburg, Sweden, psychologist; Ethel Kawin, M.A., psychologist; K. S. Lashley, Ph.D., University of Minnesota, psychologist (comparative psychology); L. L. Thurstone, Ph.D., University of Chicago, psychologist; Claude Shaw, Ph.D., University of Chicago, sociologist; John C. Weigel, administrator. The work will be carried on at the Institute for Juvenile Research, 907 South Lincoln Street, Chicago.

CORRECTION

In the abstract of the paper by Dr. John F. Fulton, "Reciprocal Innervation of Antagonistic Muscles and the Coordination of Movement," incorporated in the transactions of the Boston Society of Psychiatry and Neurology (ARCH. NEUROL. & PSYCHIAT. **16**:650 [Nov.] 1926) mention was made of the *Proceedings of the Royal Society of Medicine.* This was an error; the references given were to the *Proceedings of the Royal Society of London.*

Book Reviews

LE SYNDROME CHIASMATIQUE. (THE CHIASMAL SYNDROME). By A. FAVORY. Price, 25 francs. Pp. 184. Paris: Gaston Doin et Cie., 1926.

The book contains an extensive review of what is known concerning the anatomy of the optic chiasm, and the pathology and the clinical manifestations of disease attacking it. Few new observations are included. The author begins with clinical signs and symptoms. The visual acuity often shows less change than the visual field in diseases of the chiasm, since the fixation point is frequently spared. Careful perimetric examination is extremely important. A perimeter of the Bjerrum type is recommended, and the use of test objects of sizes varying from 1 to 10 mm. The author believes in using colored test objects also, and states that although the visual charts obtained with them are usually similar to those given by smaller test objects, yet in some cases the former will reveal defects that the latter do not disclose. It will be noted, however, that his smallest white object is larger than those used by some perimetrists in this country. The characteristic feature of the visual field is the bilaterality of the defects. The first defect is usually seen in the upper temporal sector. The fields are often asymmetric. There is no typical sparing of the macula. Rarely, there are relative scotomas for the smaller disks or for colors. Homonymous hemianopia has been seen. Binasal hemianopia is usually due to vascular or meningitic lesions situated anterior to the chiasm. Central scotomas are not infrequent, and the author suggests that the vulnerability of the macular bundle in the "toxic" group of optic neuritides may have an anatomic basis. Annular scotomas are known.

The optic disk is usually pale, but to a varying extent. Even disks apparently completely atrophic may regain some function. In inflammatory diseases of the tracts, there may be a slight edema of the nerve head. True papilledema is, however, not a part of the chiasmal syndrome (although it has been seen with hypophysial tumors).

The author lays some emphasis on the Wernicke reaction, although admitting that it is difficult to elicit and may be erroneously interpreted. He considers a roentgen-ray examination essential, but apparently does not use stereoscopic films, which are a great assistance in the interpretation of alterations of the sella.

Little space is devoted to tumors of the hypophysis, and it is mainly occupied by a review of the literature.

Four types of syphilitic lesion are recognized: a neuritic type, a meningitic type, one resulting from syphilitic lesions of the region of the tuber, and one associated with a periostitis of the floor of the skull. A neuritis of the chiasm is recognized by bilateral upper temporal quadrant defects; it is rare, and only clinical examples are reported. The bitemporal hemianopia of meningitis may be perfectly typical. The striking feature of the presumably syphilitic lesions of the hypothalamus is the adiposogenital syndrome rather than the perimetric observations in the cases quoted. The histories given to illustrate the cases of basal periostitis are not completely convincing.

Field defects suggesting lesions of the chiasm have been reported also in multiple sclerosis and in encephalitis. The war furnished a number of cases of traumatic lesions of the chiasm. Apparently, such lesions may arise long

after the trauma and may be progressive; under these circumstances one would think that the diagnosis of tumor would require careful consideration. Direct wounds of the chiasm almost invariably cause a complete and typical bitemporal field defect. Glandular symptoms have also been reported, and injury to the olfactory nerve is frequent. Injuries due to trauma to some other part of the skull usually produce an atypical sector or unilateral hemianopia, often with polyuria, etc., and injuries to contiguous cranial nerves.

The author regards it as firmly established that sinusitis may cause an optic neuritis of the typical chiasmal type. This opinion is based largely on the reports of Traquair and others, and the cases cited are certainly open to other interpretations.

Among toxic lesions of the chiasm, the author places the degeneration of the macular bundle often seen in hypophysial adenoma and the field defects observed in pregnancy. It seems perhaps more likely that this is a pressure effect, for Brouwer and Zeeman have shown that the macular bundle occupies a position in the chiasm where it could easily be pressed on by a tumor or the enlarged hypophysis of pregnancy or castration. Disturbances of vision are seen in the course of various "polyglandular" syndromes, which may be due to chiasmal lesion.

The author investigated the precise anatomic relations in three specimens hardened in situ. He found that the anterior border of the chiasm lies from 6 to 8 mm. behind the anterior edge of the sella, not at the same level, as usually stated. It may even lie in a plane corresponding to the quadrilateral plate of the sphenoid. The tract and chiasm lie at an angle of from 15 to 35 degrees. The pituitary stalk passes behind the chiasm, and indeed must curve around it. Thus, the relations between the contents of the sella and the chiasm vary in different persons. There is also considerable variation in the course of the arteries of the circle of Willis, which may come to rest on the chiasm, and may conceivably press on it. The author supposes that the chiasm may be caught between the optic recess and the infundibulum, as in a pair of forceps, or that traction by a tumor on the stalk may produce a constriction. This is a little difficult to comprehend mechanically. In some cases of tumor, the imprint of an artery has been found persisting at necropsy, as if the chiasm or tract had been strangulated by it.

Therapeutically, the author places great reliance on radiation, rather than on surgery. He attributes the first use of radium tubes in the sphenoid region in cases of tumor to Ferrei; this method has been used by Frazier for a number of years. Opotherapy is recommended for endocrine disorders.

A Bipolar Theory of Living Processes. By George W. Crile. Price, $5.00. Pp. 405. New York: The Macmillan Company, 1926.

Crile's early work on histologic changes, particularly in the nervous system in response to shock, trauma, anesthesia, etc., are more or less familiar to us all, as well as the fact that he has gone on from these studies to work in conductivity, temperature and physical chemistry. Furthermore, even though his hypotheses have not been universally accepted, still, like those of Cajal, they have furnished many with food for thought and a growing respect for the philosophic comprehension of the author.

In this new book, Crile's theory of life blossoms forth. He gathers together the results of his researches and those of others for the past twenty years, and announces his conception of a "Bipolar Theory of Living Processes." It is a work that cannot but hold the attention and gain the respect of the reader

for the sweeping character of the views expressed. The foundations on which the theory hinges are brought out in broad strokes, almost impressionistic in places. The wealth of the assembled evidence at times batters down all criticism, and yet at other times, the mere recounting of additional evidence is sufficient to stimulate the reader to doubt merely for the sake of argument. Could there be no other explanation? This also is a good thing, for it makes the reader think, something that the majority of authors take on themselves.

The book is rather loosely constructed, although in logical order, the enunciation of the theory being followed in turn by the argument, summaries of researches, summaries of certain generally accepted facts in their relation to a bipolar organism, and finally short excerpts from published papers that deal with particular angles of the problem.

To Crile, everything from the hydrogen atom to man stands in a series with regard to positive and negative charges. The physical bases of the atom and molecule, solution and colloid are more or less understood. When we come to living organisms, however, we also find two poles separated by a semipermeable membrane. This is simple enough in the unicellular animals, for here the positive pole, the acid nucleus, is separated from the negative pole, the alkaline cytoplasm, by a semipermeable lipoid membrane of almost infinitesimal thinness. This membrane, however, is of extreme importance, since it has a very high capacity for condensing electricity. Physicochemicomathematic studies by Hugo Fricke have shown that this lipoid film, the nuclear membrane, is on the order of 4 ten millionths of a centimeter in thickness, and that the combined capacity for the cells of the human body would be about that of a Leyden jar as large as a city block. It is in terms like this that Crile brings out the wonders of the body.

The multicellular organism, besides having the individual cells continually accumulating and discharging electric impulses, has a positive pole and a negative pole, taking the body as a whole. The positive pole is the nervous system, and the negative pole the liver. They are separated from one another by the intervening space with the semipermeable membranes of each organ. Conductivity and thermometric studies have shown that, in all the lower animals tested and in a few cases in man, the variations in conductivity and temperature were in opposite direction in the brain and in the liver, and furthermore, that no other organs showed as great variations in their respective directions. Various anesthetics, shock, trauma, exhaustion, sleep and drugs did not cause variations of the same sign in the liver and the brain.

Such a difference of potential is essential to life, and when the potential drops to zero, death ensues. The author cites experiments showing the effect on the cerebral cells of removal of the liver, showing that the staining properties of the cells disappear, and that from a tinctorial point of view the potential is reduced to zero.

Crile then proceeds to show how such a bipolar theory could explain the various manifestations found in nature, in the synapse, strychnine poisoning, tetanus, anesthesia, hyperthyroidism and infections; how the stepping up of potential in certain cells may cause the appropriation of nourishment to these cells and may lead to active vegetative growth rather than to differentiation. In other words, he applies the bipolar theory to the explanation of cancer. Biophysical data are then given to show that the facts are in accord with the theory. The problems of memory, reproduction and heredity are also attacked, but here the argument is not altogether convincing.

As a matter of fact, one misses the closely reasoned and carefully explained work on metabolic gradients of Child and the logical presentation of Kappers in the question of neurobiotaxis. Crile's examples are taken from widely different fields of research and serve at times to confuse the reader who is not familiar with them all. Moreover, in treating of reproduction, which, after all, is the turning point of life, he stumbles. What makes a cell divide? Crile says (p. 149): "Since the kinetic forces of the nucleus are greater than those of the cytoplasm, these repellent forces within the nucleus would finally be sufficiently strong to cause a complete division of the nuclear substance." Why? and How? These questions are not answered and probably will not be for some time to come. Yet the author calls particular attention to the attraction sphere and the spindle of mitosing cells as being visible manifestations of an electric or electromagnetic process. What is transmitted in reproduction, according to Crile, and this is one of his best points, is a "pattern of energy" which is carried out by succeeding generations of cells.

DIE CYTOARCHITEKTONIK DER HIRNRINDE DES ERWACHSENEN MENSCHEN. (THE CYTO-ARCHITECTONICS OF THE CEREBRAL CORTEX OF ADULT MAN.) By PROF. CONSTANTIN V. ECONOMO and DR. GEORG N. KOSKINAS. Price, 600 marks. Pp. 849, with 162 text illustrations and 112 photomicrographic charts. Berlin: Julius Springer.

This monumental work, ten years in the making, does credit not only to the authors but also to the publishing house financially brave enough to produce it. Since the epoch making studies of Campbell and Brodmann, very little has been added to the study of the normal cortex, except in sporadic articles, especially from the laboratory of Prof. Oskar Vogt in Berlin. The gaps left by Brodmann have been admirably filled in by this work on the normal structure of practically every area of the cortex.

The work consists of two parts: a book of 800 pages giving a description of the entire human cortex with 162 illustrations, which is practically an encyclopedia, and an atlas of 112 photomicrographs, 19 inches square. The latter are large enough to use for teaching purposes, and represent 107 cytoarchitectonic fields.

The book is divided into fourteen chapters as follows: (1) Introduction with historical sketch, considering the work of Meynert, Betz, Bevan Lewis, Hamarberg, Flechsig, Bolten, Campbell, Elliot Smith, Brodmann, Vogt, C. Jacob, and others, and then a discussion of the plan of the entire study. (2) General discussion of the cortex and its nerve cells. Here the thickness of the cortex, the brain volume and the extent of the cortex are considered, with the various cell types, cell density and arrangement. (3) Embryology. This is divided into development of the isocortex and the allocortex. (4) Peculiarities of the arrangement and the significance of the lamellar structure. (5) Divisions of the cortex into areas. Then there is a special part devoted to a discussion of each area of the cortex given as fully as possible.

Too much praise cannot be given this excellent book. There should be a copy in every laboratory in which an effort is being made to study the cortex, both normal and pathologic.

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